THE PRESENT STATUS

OF THE

PATHOLOGY

OF

CONSUMPTION AND TUBERCULOSIS.

BY

J. HILGARD TYNDALE, M.D.,

OF NEW YORK CITY.

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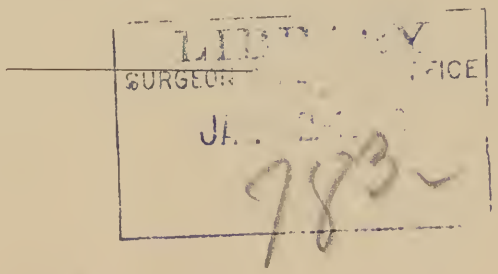

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THE following is a series of papers, written at short intervals to a fellow-practitioner in a small town. At his suggestion they are now submitted to the profession at large, with such slight alterations as the circumstances of the case seemed to call for.

J. H. T.

CONTENTS.

THE PRESENT STATUS

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Pathology of Consumption and Tuberculosis.

INTRODUCTORY.

In the present series of papers I propose giving a condensed outline of the present accepted theories in regard to the pathology of pulmonary phthisis.

Consumption has existed since time immemorial, or, at least, as far back as medical history reaches. Whether it has decreased or increased cannot be accurately determined.

Theories have been built up, only to be replaced by others; many a talent has gone to its grave, and yet it would seem as if we were still far, very far removed from the truth—at least so far as practical therapeutical results are concerned.

Lately, however, considerable light has been shed upon the subjects of phthisis and tuberculosis by a number of authors, and it is from this accumulated material that it will be my endeavor to present a *correct and distinct outline of the present status of the pathology of pulmonary phthisis, or consumption*, according to the latest accepted theories, confining my own observations to a few points, which will be duly chronicled in their proper place.

We should dismiss from our mind the idea that the pathology of phthisis will ever be brought down to represent *one single condition and its consequences.*

In order to properly comprehend upon what ground our present theories rest, it may be well to give a rapid sketch of the past history of consumption.

Consumption—decline—must needs exist where there is either

too great a waste or *a lack of supply*. Where too much is expended, where the body is reduced by discharges or secretions of any kind, we have decline, even though the supply may still be normal. The oldest pathologists have always held that consumption takes its origin from various forms of disease and various organs, and this general idea is correct to-day.

It was when autopsies became more frequent and small gray nodules were found in the lungs and digestive tract, that the theory gained ground that all consumptive processes owed their origin to the formation of nodules of tubercular substance. All consumptions were tuberculous. This was the opinion of Laënnec, who, besides the small gray nodules, described certain other larger knots, together with solid infiltrations of considerable extent, which were in nowise connected with tubercle. The first were denominated *yellow tubercle*, and considered quite distinct from the *gray* (true tubercle). Infiltrations and indurations were pronounced to be concomitants of both yellow and gray tubercle, and both were said to undergo the same processes of degeneration, by formation of caverns and leading rapidly to destruction. In other words, the division was into *tubercle proper* (miliary tubercle of present date) and *infiltrated tubercle* (infiltration generally), both subject to caseous degeneration, which process was denominated "tuberculization."

Laënnec's theory is the basis from which all else has since taken shape, and the microscope has partly laid bare the more minute structure of his two tuberculizations.

The establishing of pathological anatomy as a special branch of our science made us acquainted with the various forms of degenerations—fatty, amyloid, the proliferation of connective tissue with subsequent contraction, cirrhotic conditions. Based chiefly on the latter form, it was held that this was a decided step towards the ultimate cure of phthisis, to wit: by cicatrization of cavities. But we are forced to agree with Prof. Ruehle, of Bonn, when he says: "The fact of a phthisical patient dying of dropsy instead of consumption can never satisfy the sincere therapeutist."

Next in order of theories we have the *resemblance of consumption to inflammatory processes*, based upon the illustrations of Carwell (1838). It was demonstrated to how great an extent the smallest bronchi and the alveoli of the lungs were affected in

tuberculosis; how the changes pass into the neighboring parenchyma, the bronchi become dilated, and by dilatation finally form cavities. Again, much stress was laid upon the proliferation of connective tissue and its consequent cicatrization by induration. The fact, however, remained, that breaking down of the products of chronic inflammation was by far the most frequent result, both in the greater number of cases as well as in the extent of tissue involved; in short, that cheesy metamorphosis was the prominent characteristic. Thus originated chronic pneumonia with cheesy degeneration (caseous pneumonia). The conclusion derived from this was the recognition of tubercle in two distinct forms once more, namely: miliary tubercle (the tuberculum nodule), and the infiltrated tubercle, recognized as the result of the cheesy degeneration of inflammatory products.

It was not long thereafter that miliary tubercle was found to be a new formation (neoplasm), and since the products of inflammatory processes could not be placed with neoplasmata, it became plain that the term "tubercle" would not answer for both conditions.

In order, therefore, to determine whether tubercle and cheesy products were identical, inoculation upon animals was resorted to, and with the following result: inoculation of the products of cheesy degeneration produced both miliary tubercle and cheesy products in the inoculated animal. Right here it is well to bear in mind the possibility (and, according to late researches, the probability) of having genuine tubercle intermingled with cheesy products in the mass to be inoculated. The inoculation of miliary tubercle, however, produced miliary tubercle alone, so that, as to whether cheesy products can be brought about by tubercle direct, or as a subsequent result, has as yet not been definitely settled.*

So it would seem that thus far, at least, the identity of the two remains undisturbed, that in a general sense tubercle and caseous degeneration are convertible.

It has been demonstrated that in animals a cheesy infiltration may call forth tubercle-efflorescence, the same as in the human

* The latest investigations in regard to inoculation will be spoken of in a chapter on tuberculosis, where prominent authors will be cited to show a histological difference between miliary tubercle from a previously existing cheesy centre, and the tubercle of inoculation with an indifferent substance.

being, where general or local miliary tubercle (*e. g.*, of the meninges, meningitis basilaris) can be produced by the presence of cheesy aggregations, usually having their seat in lymphatic glands.

What has been said will suffice to make it clear to your mind that a thorough knowledge of the acute inflammatory conditions of the lung must of necessity precede an investigation into the pathology of consumption and tuberculosis. In subsequent papers, therefore, it will be my endeavor to sketch the acute lung affections (catarrhal, croupous, etc.), following them to their possible terminations, and add to these the primarily chronic inflammation of the lung (interstitial pneumonia); next treat of that chronic condition which may result from either of the foregoing, namely: Caseous pneumonia; and lastly, of tuberculosis proper, both as an acute infectious disease, as well as in its relation to caseous degeneration and to scrofulosis.

Before closing this first paper, I feel it incumbent upon me to draw attention to a few of the more salient points in the anatomical and physiological relations of the lung, so necessary to a thorough understanding of the pathology of phthisis, the more as a few points I will mention are of recent origin, and dwelt upon in the work of Buhl, of Munich, from whom I quote:

"In sifting the inflammatory conditions of the respiratory organs, you will find some of them taking place and running their characteristic course in the epithelial surface of bronchi and lung alveoli. These processes may thence be termed superficial. Other conditions manifest themselves in the walls proper of the bronchi and the adjacent actual parenchyma of the lungs themselves. Not that there is a strict division into superficial and parenchymatous inflammations, since in many processes both are more or less affected, but one of these processes is always secondary to the other."

A very important point, and one to which I desire to draw attention particularly, is the epithelial layer of the alveoli and its physiological character. Buhl, in his excellent work, while acknowledging the existence of epithelium, both in fœtal and adult age, ascribes to it the characteristics of lymphatic endothelium, spread over the inner surface of the alveoli, and not those of a continued bronchial epithelium. He proves this by drawing attention to the form and size, and the oft-asserted fact of its

being "interrupted," which is likewise, and only found, in the endothelium of serous membranes. Accordingly, bronchial epithelium passes into alveolar endothelium, as tubular epithelium is followed by endothelium in the peritoneum.

Another striking proof is found in the experiments of Sigorsky (*Centralblatt*, 1870, No. 52), showing the existence of a network of lymphatic vessels, consisting of canaliculi and stellate knots uniting them in the alveolar walls, which knots communicate with the volume of the alveoli by means of minute openings. It would appear proper, therefore, to look upon the alveolar pouches as wide lymph-spaces, filled with air and lined by endothelium, which endothelium is wanting in the lymphatic network of the alveolar walls. These relations are of importance for the exchange of gases in respiration, since the inspired air meets with no absolute resistance within the walls of the air-cells, which would not merely permit of diffusion of gases through the capillary walls, but of a direct entrance into the open lymphatic network of the alveoli.

Lastly, attention has been called by several authors to the analogy between the alveolar epithelium and the endothelium of the lymphatics, more especially as regards their products in pathological processes. It is well to remember that authors, speaking of superficial inflammations, thereby designate such forms, the products of which are chiefly generated in the epithelium or pass over its surface; while the products of parenchymatous inflammations are interstitial and peribronchial.

The vascular system of the respiratory organs, likewise, calls for a moment's attention. It is well never to lose sight of the fact, that the arteries proper of the lungs are not the only ones, but that the bronchial arteries likewise may play an important part. The bronchial arteries have a share in the capillaries of the pulmonary organs, and therefore take a part in the disturbances of nutrition of lung tissue. The bronchial arteries run an interstitial, interlobular course, finally branching out on the pleuræ, while the arteries of the lung proper form the great bulk of capillaries in the alveoli.

It follows, therefore, as a general rule, that peribronchial inflammations and parenchymatous infiltrations of the lungs are within the territory of the bronchial arteries, whereas the superficial inflammatory conditions (exclusive of the bronchi) are with-

in the territory of the pulmonary arteries. It further explains the involving of the pleura and bronchi in original maladies of lung parenchyma, as well as the possibility of an extension of bronchial or pleuritic disease upon lung parenchyma, and on the other hand the well established regularity in the appearance and character of one or the other of the diseases named, to wit: Primary disease of lung parenchyma is more diffused, more continuous in accordance with the capillary distribution; in other words, it is lobar; whereas, primary disease of the bronchi always leaves certain branches free, and even when spreading to the lung parenchyma will, at first, be lobular, or even remain so. In other words, diseased lobules are contiguous to, and surrounded by, relatively healthy ones.

The same thing may be said of thromboses and emboli. Ordinary thromboses occur in the usually dilated capillaries, which are fed by the arteries of the lungs, and create lobar trouble, the so-called pyæmic plugs (emboli) in the finer arteries of the lungs are, though small, distinctly lobar as regards their seat and extent.

Intermediate between the lobar and lobular form, we place such disorders which originate in the lymphatics. Of these, Prof. Buhl says: "As the finest ramifications of the lymphatics follow the course of the bronchial arteries in the alveolar walls, running their course interlobular, interstitial and peribronchial, congregating at length partly in the subserous pleural tissue, partly in the lobes of the lung, so is their pathological progress either continuous and diffused, or confined to lesser spaces, to mere points; or thirdly, both conditions may exist simultaneously and a doubt may arise as to whether they should be called lobar or lobular."

There remains still another point for examination. It is that an acute pulmonary trouble will present itself quite different from a chronic one. Inflammations, either superficial or parenchymatous, whether lobar or lobular, will present quite different features when of acute than when of chronic origin. If acute, of recent and sudden origin when examined, they differ widely from chronic inflammations, whose chief characteristics are degenerations of great extent when superficial. On the other hand they are characterized by proliferation of connective tissue, cicatrization, etc., when such chronic inflammations are parenchymatous, interstitial or peribronchial.

In either case, it will be well to remember, that in an inflammatory process, whether acute or chronic, where secretions are not expelled, but remain in the respiratory passages, as well as when tissue is being disintegrated or destroyed, the contact of atmospheric air (peculiar to the respiratory organs) exercises a disintegrating effect upon the infiltrations, in which the whole body participates. This is seen in the shape of fungi, which enter the blood and lymphatic vessels through the decomposing tissue.

CATARRHAL PNEUMONIA.

You will remember I stated in my first article, that a catarrhal process of the lung tissue proper (of the alveoli) was an impossibility, on account of certain anatomical conditions. Still the term catarrhal pneumonia has become so fixed in our nomenclature, as to mislead many practitioners in regard to the pathological conditions passing under that name.

French physicians first drew a line of distinction between catarrhal pneumonia and croupous pneumonia. Among Germans, Ziemssen has furnished the best and most comprehensive view of the subject in his "Pleurisy and Pneumonia in Infancy."

Niemeyer, in his chapter on catarrhal pneumonia, opens by saying that since no mucous membrane with mucous follicles existed in the alveoli, it was in reality a catarrh of the finer ramifications of the bronchi. Notwithstanding the extensive researches of Bartels, Rokitansky and others, the pathology of catarrhal pneumonia has been all but comprehended. According to Rokitansky the minute *bronchi and the final termini of the bronchi* are the seat of an intense inflammatory process. Adding to this dictum the result of the investigations of other authors, we may say: It is an *acute* disease, attacking the lower lobes, beginning at their borders and subsequently passing forward and upward.

Juergensen, on the contrary (see Ziemssen's Cyclopœdia), insists that in order to comprehend the etiology of catarrhal pneumonia, it is necessary to limit the definition thus: "Catarrhal pneumonia is always a secondary process and never has its primary origin in the alveoli. As a rule, it is preceded by inflammation of the bronchial mucous membrane. The only exception to this rule is in the inhalation of strong irritants, such as chlorine, where the effect may be simultaneously produced in bronchial and alveolar mucous membrane." It will occur at once, that a question of etiology of such importance should not be left in doubt, if possible, especially since it must needs have a direct bearing upon the possibility of its being a basis for phthisis. These points will be touched upon in their proper place.

All observations tend to establish that catarrhal pneumonia is most frequent in children under five years of age.

I will here positively assert, that *catarrhal pneumonia is the only form of inflammation of the lungs ever occurring before the age of puberty.* Croupous pneumonia never occurs before this age. This may seem heresy. Much as I regret it, the extent of the subject will not allow me to enter upon this question.

Acute catarrh of the *lesser branches, as well as of the most minute termini of the bronchi, is,* like all catarrhs, *superficial, accompanied by œdematous swelling and redness of the mucous membrane.* The immediate result of the foregoing is excessive secretion of mucus, largely diluted with serum, occasionally mixed with blood corpuscles and liquor sanguinis. In time, with the diminution of serum, the secretion becomes muco-purulent and purulent.

This short outline of the pathology of catarrhal pneumonia will show the propriety of denominating this disease by the name of *broncho-pneumonia,* according to Niemeyer and others. There is still another term for this disorder—*capillary bronchitis.* It is the one mostly used in this country for defining the progress of bronchial catarrh in the bronchial ramifications or capillaries.

Let us now pass to the actual pathological processes of catarrhal pneumonia. That the alveolar walls are a continuation of the bronchial ramifications, has been dwelt upon by Oppolzer, Niemeyer and others, both of whom, strengthened by Buhl, have taken occasion to dwell upon the fact, that this continuation, however, involves a complete change in the character of the alveolar lining. Bronchial mucous membrane is composed of several layers, has alveolar membrane, which, as has been previously stated, is very much like lymphatic endothelium, has pavement epithelium, and consists of a single layer.

In accordance with this anatomical arrangement will be found the pathological conditions. It is easy of comprehension, that an actual catarrh of the middle and finer bronchi, and the clogging up of these with mucus and pus, must needs involve the alveoli in a decidedly modified degree. I will first give an exact outline of the accepted pathological conditions, and then pass to the causes and their effects, such as are closely allied to catarrhal pneumonia.

In examining an acutely diseased lung, we find different parts

of the lung in different conditions. We find, 1st, lobules, which upon section present a smoothly cut surface, not granular as in croupous pneumonia. Corresponding to the lobule, this smooth surface is found to be part of a well solidified, condensed deposit, for the most part situated in the periphery of the lung. On pressure, a fluid, at first bloody, then of pale gray color, escapes, mixed with hyperplastic epithelial cells, part of which are already undergoing fatty degeneration. The chief component part of the infiltration, the gray fluid (for *epithelium* is secondary in quantity) is a *secretion of thick mucus, very rich in pus corpuscles.*

2d. Parts in which the quantity of blood is greater than in the foregoing, the pleuræ even showing ecchymotic spots. On section, serum mixed with blood and air bubbles escapes freely. This is acute œdema of the lung.

3d. In other lobules the air-cells appear distended with air, quite pale, and showing vascularity in their periphery only, as if enclosed by blood-vessels (local emphysema).

4th. On the borders of the lung and toward the root, the alveoli are collapsed, retracted, entirely or nearly emptied of air, and of bluish red color. These are atelectatic parts.

Of the above four conditions found in lobules, only the first (filling up of the lobules with mucus and pus) is the immediate and actual result of catarrhal pneumonia. Acute œdema, local emphysema and atelectasis are merely concomitant conditions, the same as accompany other inflammations. Again, all four conditions may alternate with perfectly healthy tissue, thus showing the *lobular* character of the existing inflammation.

It will naturally be concluded from the above, that, since the mucus and purulent products are found in the alveoli, they must be products of their structures. This may be partly true with respect to the pus, since the alveolar capillaries may permit of migration of white (pus) corpuscles, as well as other corpuscles. As it is pretty well established that alveolar epithelium does not produce mucus, the greater part, if not all of the secreted product must have originated in the smallest bronchi and have been transferred by respiratory motion and *gravitation* to the alveoli. That the alveoli are at the same time in a state of inflammatory irritation, may be allowed.

Catarrhal pneumonia should, therefore, be defined as: A CAPILLARY BRONCHITIS (SO-CALLED BRONCHIOLITIS), IN WHICH THE

MUCOUS AND PURULENT SECRETIONS OF THE BRONCHI ARE TRANSFERRED BY RESPIRATION AND GRAVITATION TO A NUMBER OF ALVEOLAR LOBULI, CAUSING THE LUNG TO PARTICIPATE BY COLLATERAL ŒDEMA, LOCAL EMPHYSEMA, ATELECTASIS, AND HYPERÆMIA.

It is not my purpose to dwell at large upon prognosis, etc. As stated in the first article, I will chiefly confine myself to pathological conditions.

A favorable termination is due to the above-mentioned fact, *i. e.*, the non-participation (at least in a direct manner) of the true parenchyma in the inflammatory process. The normal condition is regained by fatty degeneration of the products of the inflammation, together with their expectoration through the bronchi, accompanied by the absorption of the pus-corpuscles (white blood-corpuscles) still remaining. Renewed active circulation in the capillaries, reabsorption of serous infiltration into the lung parenchyma and bronchial mucous membrane, restitution of local emphysema, expansion of collapsed parts by the entrance of atmospheric air and regeneration of the lost epithelial cells, take place.

Thus I have merely sketched the favorable termination of this kind of pneumonia, in order to transfer your attention to the changes which occur when absorption, etc., does not supervene, and which changes pave the way to pulmonary consumption.

The factors which should govern our prognosis have been well condensed by Juergensen, as follows:

1st. The more acutely a catarrhal pneumonia runs its course, the less the danger of an unfavorable termination.

2d. The younger the patient in number of years preceding the age of puberty, the greater the danger to life.

3d. The danger to life is in exact proportion to the extent of the pneumonic process.

4th. Any reduction in the general resisting power of the patient increases the danger to his life.

It is not my province to dwell upon other possible terminations, for example gangrene, as that would not be appropriate to our subject, which is to confine ourselves to such conditions as lead to pulmonary consumption or phthisis.

Where the inflammation passes into the chronic condition, as frequently happens to debilitated persons, quite small sections of the parts involved may linger behind others entering upon resolution, and thus pass into the chronic stage.

In speaking of the restitution of local emphysema, as well as of collapsed parts, it must be borne in mind that, inasmuch as clogged-up bronchi exist in such parts, residues may readily be left behind. This is true of incapsulated epithelium, as well as of the mucous and purulent secretions, all of which then tend to *caseous degeneration and pigmentary degeneration.*

Authorities differ in regard to the frequency of cheesy degeneration of these products. Niemeyer states that it occurs very frequently. Bartels and Ziemssen have seen each two cases only where non-restitution resulted in cheesy products and the subsequent development of tubercle from that centre. Buhl places this occurrence among the rare events. As a substantiating proof he calls attention to the fact that cheesy degeneration is chiefly found in the upper lobes and their apices, while, as above related, the products of catarrhal pneumonia are found in the lower lobes. Then the catarrhal products only clog up the finer bronchi, are not traversed by lung-tissue proper, nor is any of it included in the cheesy degeneration, as is nearly always the case in true caseous degeneration. Buhl goes on to say, that "the bronchiolus encasing the cheesy mass is dilated and thickened, forming, as it were, a fibrous capsule for the mass."

Mark now that this process of incapsulation of the cheesy mass is not one of the effects of catarrhal pneumonia (although the products of the cheesy mass itself is), but the result of the activity displayed by the surrounding parts (connective-tissue vessels), irritated by the existing inflammatory products. This irritation *is not of a very acute character*, and tends to the formation and subsequent contraction of connective tissue. This is true chronic peribronchitis, of which more will be said in a future article.

Next let us dwell a short time upon a point in connection with the origin of lobular cheesy masses, which are produced by the wandering of catarrhal secretions from the lesser bronchi into the alveoli. The same process, as asserted by some writers, is brought about by the entrance of *foreign bodies* into the alveoli. In the category of foreign bodies, they include extravasated blood, croupous and diphtheritic membranes. It is claimed that by inspiration they are driven into the smallest bronchi and lung alveoli, and there, by continued irritation, give rise to superficial epithelial disturbances.

To these products may be added the pus of purulent bronchitis and that from cancers, as well as all products of ulceration (such as epithelium from cancer of the lip), particles of cartilage from necrosis of the larynx, or particles, in fact, from any destructive process.

Let us dispose of these pathological products before considering such as are introduced from without. It is well, while allowing for their ability to cause superficial inflammation, to discriminate between each one of these products. It should be borne in mind that extravasated blood is often coughed up within the next few days succeeding a hemorrhage, while the blood that remains is apt to become purulent so rapidly as to suggest the probability of its expectoration before its presence could have caused any great damage. Products of a *chronic* character (pus from cancers), continually supplied for a long time, are more apt to degenerate.

With regard to croup and diphtheria, which affect the laryngeal and tracheal mucous membrane, opinions differ.*

Foreign bodies introduced from without should be briefly touched upon. The inhalation of dust has of late received considerable attention. Ever since Prof. Tyndall's lecture on "Dust and Disease," and Zenker's essay on "Pneumonokonioses," this subject has created lively interest. The presence, under certain conditions of the atmosphere, of atoms of lime, silicates, carbon, various metals, has caused Zenker to base thereupon his theory of "foreign body pneumonia." These atoms have been found to exist in the alveoli. Of course, in the majority of cases their presence is harmless, but I can readily imagine that in a broken-down constitution, or in cases in which the innervation of the alveoli is disturbed, pathological processes may supervene.

Mention is made of still another ending of catarrhal pneumonia, which is a rare one. I refer to atrophy of the alveoli and to bronchial dilatation. This condition supervenes mostly in the

* Prof. Jacobi, who is the best authority on this subject, tells me that the decomposed material is carried downward by the force of inspiration, and broncho-pneumonia may result from atelectasis. "Sometimes embolism results from thrombi in small veins, either near the larynx in croup, or in other parts from debilitated circulation, or by portions of a heart-clot torn away." Extension by contiguity, he says, is found to reach no farther than the second ramifications of the bronchial tubes.

aged, though it is well to bear in mind the possibility of its occurring in infants, who are the chief victims of catarrhal pneumonia. This is said to occur both in the upper and lower lobes. The bronchi, even in their earlier ramifications, become cylindrical and varicose, with rapidly succeeding dilatations. The mucous membrane remains intact. The alveolar structure has disappeared; emphysema supervenes. The alveolar epithelium is either fattily degenerated, or has entirely disappeared; obliterated capillaries and lymphatics take their place This latter fact demonstrates that the original process was one of obliteration (by pressure) of the capillaries. Therefore, this is true atrophy.

Alveolar atrophy does not lead to phthisis, since it may properly be called a curative process—an attempt to repair, and, when acquired in childhood, often carried to a green old age.

CROUPOUS PNEUMONIA.

The term "croupous," as you know, is used in connection with a number of diseases, as of the larynx, lungs, and kidneys. Flint describes all croupous affections as being characterized by an exudation of lymph. It is well to have in mind that this exudation is *upon* the surface of membranes in contradistinction to diphtheritic exudation which takes place within the tissue itself. German authorities, therefore, invaribly define croupous inflammations as characterized by an exudation, which is rich in fibrine, upon the surface of mucous membrane. Latterly the identity between croupous and diphtheritic exudations has been sought to be established, at least so far as laryngeal troubles are concerned. But to dwell upon this point would take us too far from our real subject.

Again, one of the pregnant questions of the day is, whether or not croupous pneumonia is an *acute infectious disease*, running as it does a decidedly typical course.

Juergensen makes the following declaration: "Croupous pneumonia is a disease affecting the system generally, not a particular locality. Inflammation of the lung is merely a prominent symptom. The phenomena of the disease cannot be accounted for by the local conditions."

Briefly let me give the evidence in favor of the above, as gathered from this and other sources, and from the etiology, anatomy, and pathology of the disease:

1st. The seasons at which pneumonia occurs, differ from those of inflammatory conditions of the lung. It ranks fairly as an endemic, occurring with a certain regularity at certain seasons of the year.

2d. During the whole course of the disease there is no constant relation between the degrees of fever and the local symptoms; consequently the length of the fever is not to be credited to the local conditions.

3d. The decidedly typical course.

4th. Inflammation of the lung accompanying typhoid fever and other infectious diseases is not croupous pneumonia; there-

fore croupous pneumonia is never a secondary, a consecutive inflammation.

5th. Death is often due to paralysis of the heart, as it occurs in other acute infectious diseases, where the quantity of infection can be estimated in a measure by its effect upon the innervation of the heart.

6th. Attempts to abort, to cut short the course of croupous pneumonia, have tended only to enfeeble the already weak powers of resistance of the patient. In view of the numerous discussions of late in regard to this point, I regret that we cannot now go further into this.

The inflammatory process itself is one affecting the alveoli and bronchi, in the course of which an exudation *rich* in fibrine is poured out upon the surface of the mucous membrane and then *coagulates*. No one has ever attempted to establish the existence of a croupous pneumonia running a chronic course, and I wish this point especially to be borne in mind, when I come to speak of the possible terminations of croupous pneumonia and their results.

The croupous process is a truly superficial one; indeed, quite as much as the catarrhal process. While in catarrh we have transudation of serum from the blood, in croup there is *added* an abundance of fibrine. You will find it stated by most authorities that the exudation *raises* the epithelial layer, thus doing away with the latter. This is erroneous. It has been established long ago that the fibrine *passes over* the epithelium, as any ordinary secretion, and the epithelial layer is lost by subsequent degeneration. Niemeyer had always held that an exudation, rich in fibrine and quick to coagulate, is deposited upon the surface, enclosing within itself the normal epithelium and newly-formed cells. The fact is, the epithelial layer covered by the fibrinous exudation undergoes no other changes save œdematous swelling, opacity, and incipient fatty or mucous degeneration.

Passing to the pathology proper of croupous pneumonia, we find it almost invariably involving the greater portion of a lung, beginning mostly at the root, involving the lower lobes, and thence passing to the upper lobes. And right here is a chief distinction between the characteristics of croupous and catarrhal pneumonia. Both are superficial. In the catarrhal form, however (*vide* my second article), the process is one derived from the

smaller bronchi, and hence *only* lobular, while croupous pneumonia, involving the lung parenchyma proper, is *diffused* as well as lobar, involving the bronchi only as a secondary process.

Croupous pneumonia has three distinct stages. It will be necessary, for completeness' sake, to dwell upon each of these stages, in order to arrive at safe conclusions regarding the final termination.

In the first stage, that of *inflammatory determination or engorgement*, the lung parenchyma is of dark red color, is heavy and harder, and has lost its elasticity. On section, the cut surfaces present a dark appearance, and blood flows in abundance, together with serous fluid more or less frothy. In speaking of the possibility of considering croupous pneumonia as an exaggerated catarrh, Buhl says: "When we see in catarrh serous exudation, superadded to the normal secretion of the mucous membrane, the same addition is present in croup of the pulmonary alveoli in the shape of gas and watery vapor, changing at once into perfect lymph. Looking at it from this standpoint, the inflammatory engorgement of the lungs, characterized by transudation of bloody serum, is analogous to catarrh of the bronchial mucous membrane; and if this goes on gradually, the exudation contains more and more fibrine, the process is aggravated into croup." I quote the above merely as an individual opinion. It is neither an accepted nor an established fact.

The passage from the state of engorgement to that of hepatization is always of short duration, commonly in one day. The alveoli and bronchi are now filled with coagulated fibrine, mixed with blood (giving it the red color); the air has disappeared from the alveoli. Hence the lung is solidified, and the characteristics of croupous pneumonia are established. The cut surfaces present a granular appearance, most plainly observable in the larger alveoli. These granulations are small, firm props of coagulated fibrine. Flint says they are composed of amorphous fibrine or lymph in a granular form, epithelium, fatty granules, some blood-disks and leucocytes. These coagulated alveolar contents press upon the parenchyma, infiltrated with serum (œdematous), and this pressure is what causes the croup-props to bulge out as granular matter, and they are in this shape true mouldings of the alveoli and infundibula.

In this connection it is well to point out another distinction

between croupous and catarrhal pneumonia. As the whole lobe of the lung has materially increased in weight and volume, the pleura covering it is likewise tense and œdematous, and likewise becomes covered with transuded fibrine. Serous effusion also supervenes. This form of pleuritis is peculiar to croupous pneumonia, and totally wanting in the catarrhal form.

In the further course of the disease the red color of hepatization gives place to a gray or yellowish appearance, the lung texture remaining the same. I think this is due to the usual *changes in the hæmatin* of the red blood-corpuscles, as commonly seen in superficial ecchymoses, though I fail to find this stated by any author. This intermediate discoloration is sometimes denominated *gray and yellow hepatization.*

If *resolution* supervenes (after the seven days of hepatization), this is effected by fatty degeneration of fibrine and young cells, possibly also of mucous degeneration. There is softening, expulsion, as well as reabsorption. Most authorities allow that these processes go on during restitution in all parts of the diseased tissue simultaneously. Buhl, however, very ingeniously demonstrates the following facts: the *alveolar epithelium* takes the lead in fatty degeneration, becoming granular, and by this process the croupous props are detached and raised from the inner alveolar walls. The *bronchial vibrillated epithelium*, on the contrary, largely undergoes mucous degeneration; likewise the croup-props become detached and softened. The quantity of mucus formed in the smaller bronchi is always large. Now in order to expedite excretion, there is a necessary vibratory motion, a narrowing and widening of air-passages, in inspiration and expiration, and sound elasticity of the lung parenchyma. All these conditions are rendered nugatory by dense hepatization. The first step in actual degeneration is an *active cell proliferation* below the detached alveolar and bronchial epithelium. It is not until this condition is fulfilled, that vibratory motion can be possible, and this is confined to the bronchi. In order to expectorate the alveolar portion of the deposit, the factors of forcible contractility of the air-passages, as well as elasticity of the lung parenchyma, are required; yet these forces have been and will remain paralyzed for some time to come. Moreover, the sputa of pneumonia are catarrhal products of the bronchi, mixed with fibrine and reddened by the admixture of red cor-

puscles (rusty colored sputa), all derived from the bronchial mucous membrane almost exclusively. The deduction from the above is, that THE WHOLE OF THE HEPATIZED LUNG TISSUE PROPER UNDERGOES RESOLUTION ALMOST ENTIRELY BY REABSORPTION OF THE SOFTENED ALVEOLAR PROPS ON THE SPOT. This fact is important in considering further terminations of croupous pneumonia.

Resolution, when it takes place, is always complete. This proves croupous pneumonia to be a superficial inflammation only, spending its force upon the alveolar epithelium, but not penetrating into the lymphatics. Keep this fact in mind.

On the other hand, the process may pass into the third stage of destruction, so-called gray hepatization or purulent infiltration, chiefly caused by lack of the general powers of resistance and weakness of the heart's action. The pathological anatomy of these changes does not differ from those of resolution, save in that, on account of the enfeebled powers of resistance, the regenerative processes do not keep pace with those of the degenerative, or the former may be even wanting altogether. The volume and weight of the affected part remains unchanged. The granulations (croupous props) are now readily pressed out and disappear. The parenchyma remains not only anæmic and devoid of air, but is much softened, breaking down on slight pressure. Reddish gray thick pus flows freely over the cut surface, and may be readily pressed out in large quantities. But the minute structure of the lung is still unchanged, and at this stage a late resolution is still possible. Death usually ensues (as late as the tenth day), but in a few cases a small amount of surplus vitality causes the products to be absorbed, as in the hepatized condition. This, of course, is immediatly consequent upon the disappearance of the croupous props. But if absorption fails, the formation of pus (as above stated), true purulent infiltration, will lead to death. An important point in this connection is the presence of pus-corpuscles in the lung structure itself. The pleura takes part in this purulent infiltration and its effusion, consequently becomes purulent. Remember the point, in the beginning of this paper, viz.: the truly superficial character of croupous pneumonia, wherein the parenchyma was merely *œdematous*, and now by its *purulent infiltration* forms, as it were, a bridge to *interstitial* forms of inflammation, and this is in itself sufficient to lead to the formation of *abscess of the lung*. Abscess of the lung may

occur during life, but then it may be looked upon as a decidedly slow and localized process, taking weeks for its formation, since rapid infiltration would kill rapidly. Moreover, in order to form a perfect abscess, the neighboring parts must be undergoing resolution and must be healing.

I find it necessary to dwell for a few moments upon the possible ending in pulmonary gangrene. This occurs in croupous pneumonia (it may occur suddenly by thrombosis, etc.), in the stage of red hepatization. Stagnation and numerous thrombi in the branches of the bronchial arteries, which carry on the nutrition of the lung, causes the flow of blood to be cut off from the inflamed parts. Cessation of nutrition and the constant presence of atmospheric air are the factors for mortification, which involves both lung structure and the infiltration. But as it may be set down as a rule that anæmia from thrombosis is nearly always a localized process in the different organs (*e. g.*, the brain), so it is the case with the lung. The same condition and action of the neighboring parts, as just spoken of in lung abscess, here supervene. Demarcation, granulation, and throwing off of the mortified part, and indeed in both abscess and gangrene of the lung, the favorable termination consists of a loss of substance—in short, a *cavern*. Only in case of exceptionally small loss of substance and consequent caverns of the size of a pea or a trifle larger, may cicatrization and complete closure occur. This is, no doubt, the only condition under which cirrhosis of the lung (induration) occurs as a sequel to croupous pneumonia, though some authors mention diffuse proliferation of connective tissue and cirrhosis as a sequel. Of this there is no proof.

Lastly, I return again to the important question of the possibility of croupous pneumonia leading directly to pulmonary phthisis. It will be remembered that my quotations from authors, at the beginning of this paper, are to the effect that croupous pneumonia was in itself strictly superficial. If you have closely followed the pathological anatomy of all possible terminations, to wit: gray hepatization, purulent infiltration, abscess and gangrene, it will have become apparent that *one* of two results *alone* is possible, viz.: resolution and absorption, or destruction by pyæmia, or the formation of a cavern (in abscess and gangrene). The formation of a cavity, it should be remembered, is not primarily the effect or a product of croupous pneumonia, for

these products have by this time all disappeared. These facts would seem to effectually remove any doubts as to the impossibility of a termination in caseous pneumonia; and yet, as far as I am able to ascertain, almost the majority of authors state it so. Niemeyer even mentions caseous pneumonia as of quite frequent occurrence in this connection. He says, that when the extravasated fibrine has undergone fatty degeneration, and this mass is not properly supplied with serum by the alveolar walls, the fatty mass will dry and become changed into a more or less firm, yellow, cheesy mass.

Finally, let me tersely recapitulate and condense the opinions of the present day in regard to the possibility of products of croupous pneumonia leading to phthisis, that you may not get confused in regard to it.

Remember, always, that a rapidly destructive process, such as fatal terminations of lung inflammations (gangrene, abscess), bear no relation to phthisis. Bear this well-established fact in mind: THAT IN ORDER THAT AN INFLAMMATORY PROCESS SHOULD LEAD TO CONSUMPTION, ITS PRODUCTS MUST HAVE A REMNANT, AND THIS REMNANT MUST UNDERGO CHEESY DEGENERATION.

Based upon this, the only questions to be answered in relation to croupous pneumonia are: 1st. Do any remnants ever remain? 2d. Do these remnants undergo cheesy degeneration? In regard to the first question, we have exhausted the subject throughout this paper; yet, once more, I condense the views of prominent observers. They are as follows: Buhl denies all possibility of a remnant, and he has many supporters. Juergensen, Rindfleisch, and Ruehle have taken pains to oppose this view. The views of all, however, coincide in this, viz.: even if no distinct remnant remain, the inflamed parenchyma may be, to a greater or less extent, a starting-point for limited development and contraction of connective tissue (cirrhosis).

Again, a number of Buhl's cases of desquamative pneumonias may have been croupous, or croupous cases of the other authorities may have been desquamative, as they are clinically hard to separate.

And, thirdly, the possibility is held out that a desquamative process might follow directly in the lead of a croupous. Now all acknowledge (Buhl included) that cirrhotic conditions and desquamative pneumonia are prone to cheesily degenerate.

The second question answers itself. If remnants remain, they can readily undergo cheesy degeneration, especially while the patient is still in a debilitated condition.

Again: If croupous pneumonia is an acute *infectious* disease and never an accompaniment of other diseases, never *secondary* or consecutive, no remnant can remain for cheesy degeneration.

If, on the other hand, as an ordinary inflammatory disease it runs no typical course, and if desquamative pneumonia as an independent disease is a myth (as asserted by Rindfleisch), or, if existing, it may follow in the wake of croupous pneumonia, the conditions for the remaining of remnants are given and their cheesy degeneration a natural sequence.

DESQUAMATIVE PNEUMONIA.

The name of "desquamative pneumonia" will no doubt sound strange to you. I will introduce it by stating that its existence, as a separate form of lung trouble, was first mentioned by Prof. Buhl, of Munich (see Henle and Pfeufer's Journal, series viii., 1856). While its existence as a distinct and separate disease has been at times tacitly acknowledged by others, Buhl alone, as far as I can ascertain, has furnished the literature of pathology and pathological anatomy. In the present paper on desquamative pneumonia, I can only endeavor to collect his thoughts and reproduce them in condensed form.

Desquamative pneumonia occurs in two grades and modifications, each one of which will be separately considered.

The *first* variety, and the one of lowest grade, is that which occurs as an *accompaniment to severe general diseases*, and is therefore denominated by Buhl as "*consecutive desquamative pneumonia;*" it is chiefly an accompaniment of acute infectious diseases, but the introduction of any inorganic poison into the body, as pyæmia, etc., forms the basis of this first variety of desquamative pneumonia. The pathological changes in other organs, in connection with general diseases, are spoken of as parenchymatous inflammations, such as parenchymatous nephritis, hepatitis; and it would seem rational to allow the existence of parenchymatous pneumonia, though this term is not now in vogue. In consulting other authors, it will be found that they speak merely of hyperæmia, hypostatic congestion and œdema of the lungs in connection with infectious diseases; but the pathological anatomy of the process seems to have often been supposed to be that of croupous pneumonia. I find it stated, with strange uniformity, that pneumonia accompanying typhoid fever is croupous, and originates under the same influences as does catarrh of the air-passages in measles, without any reference to the existing pathological condition, which corresponds to our desquamative form and differs materially from croupous and catarrhal pneumonia.

Consecutive desquamative pneumonia (alike desquamative

nephritis) occurs *bilaterally*, is *diffuse*, consequently *lobar*, and *affects the bronchi secondarily only*. The lungs are enlarged, gorged with blood, minute extravasations exist in the centre of the parenchyma, as well as subpleural; do not collapse when removed, notwithstanding they still contain air. Foamy serum flows from the cut surface, and very minute excrescences are seen. The tissue is soft and readily torn. The microscope here becomes indispensable and reveals epithelium of the lung alveoli in great quantity; they are bloated and rounded off, filled with fine granules. This is proof positive of the changed cells raising themselves from their stratum and from each other; in other words, desquamating, analogous to parenchymatous nephritis. Hence the name.

Catarrhal and croupous pneumonia are characterized by changes in secretions, while here SEROUS INFILTRATION OF THE PARENCHYMA IS THE CAUSE OF THE DESQUAMATION OF THE EPITHELIUM.

Pus-corpuscles are not present. Pleurisy is wanting. Where death does not ensue in consequence of the acute infectious disorder and the accompanying desquamative pneumonia has not reached too high a grade, resolution takes place by transitory fatty degeneration of the detached epithelium and regeneration of the same, as hyperæmia and swelling disappear. This is analogous to what occurs in the liver and kidneys.

Death, however, may not be caused by the general disease, and disease of the lung may have been of so high a grade as to require time for the ultimate removal of its product. In such cases we are dealing with a chronic fatty degeneration, of which more will be mentioned hereafter.

Buhl insists (see Virchow's Archives, XII., 1857) that another termination of consecutive desquamative pneumonia is in *acute* atrophy of the lung, formally disposed of as a result of hypostatic or catarrhal pneumonia. It would take us too far away from the subject-matter proper to dwell upon this condition further than to say that he refers to the lung in an atelectatic condition.

The *second* form of this disease is that which is denominated *genuine* desquamative pneumonia. It holds the same relation to the foregoing consecutive form as does Bright's disease to the kidney affection accompanying general diseases—albuminuria.

It is therefore to be regarded as an independent, a primitive inflammation, or, in other words, the localized expression in the lungs of a general disease.

When confined to one lobe, it is usually the *upper* one. Again, it may involve the whole lung, when the upper lobe will prove to be the one most affected. The inflammatory process is almost invariably further developed in the upper portions and its downward course can be readily noted.

The pathological anatomy of such a lung presents the following picture in cases which have lasted from six to eight weeks: volume and weight of the diseased lung are augmented; the surface is smooth, of dull lustre. The pleura is œdematous, with sparse ecchymotic spots. The lung does not collapse either by opening the thorax or upon incision; its elasticity appears to be entirely wanting. Nevertheless, it is quite frangible. On section we readily discover the lobar (diffused) character of the inflammation. There is also a granular surface, as in croupous pneumonia. In croupous pneumonia, however, the granulations are filamentous plugs partly passed out of the alveolar spaces. Here, on the contrary, we have the granulations formed by the *thickened interalveolar parenchyma*, rendered firm by the great œdema and bound down by newly-formed connective tissue. The parts are rich in blood, and pigment in granular form is present in quantity corresponding to the duration of the disease. On account of the parenchyma being firmly bound down, only small quantities of gelatinous fluid can be scraped off from the surface; this fluid is freely mixed with cellular elements. The cellular elements serve to distinguish this form from consecutive desquamative pneumonia, in which œdema alone is present.

The exudation of genuine desquamative pneumonia is a plastic exudation.

The microscope reveals detached epithelium in profusion; these are from the alveoli as well as the finer bronchi, which they fill. These epithelial cells are undergoing the process of fatty degeneration; neither pus nor mucus is present. Let me repeat: the plastic exudation consists of epithelium in profusion, filling alveoli and finer bronchi and undergoing fatty degeneration. This exudation is bound down by newly-formed connective tissue.

Clinically, this is of great importance in connection with the

sputa, which contain alveolar epithelium in large quantities, something not occurring in catarrhal or croupous pneumonia.

Another characteristic of the epithelium is the proliferation of nuclei and younger cells, denoting active regeneration and plastic activity from the beginning of the disease. In croupous pneumonia, it will be remembered, regeneration is the closing act.

The four chief characteristics, viz.: firmness of parenchyma, pigmentation, proliferation of connective tissue, and a profusion of epithelium, serve to stamp genuine desquamative pneumonia as a *parenchymatous inflammation*, far in excess of the consecutive form.

Buhl points out (page 53) that genuine desquamative pneumonia serves to show the connection and analogy between connective-tissue corpuscles, lymph-endothelium and alveolar epithelium, since the desquamation of alveolar epithelium cannot be traced to œdema of the alveolar walls alone, but must be regarded as proliferation and degeneration of tissue elements of one species.

Though likely to end in death, complete recovery is possible, at least as often as in Bright's disease. The great majority of cases, however, are subject to frequent recurrence and gradual continuance of the same processes, thus bringing about permanent and irremediable conditions. And this brings us to our chief point, namely: its termination into consumption. The form in which this is accomplished is that of *chronic fatty degeneration*, and the process is simply the continuation of the acute form: proliferation and fatty degeneration of epithelium becoming chronic. The clinical appearances are from thence those of phthisis.

In such cases general anæmia of course supervenes, which accounts for the lung being found *quite pale*. It is of light slate color, owing to absorption of the pigment, and dotted with minute white specks—alveolar spaces filled with fattily degenerated epithelium.

I now wish to draw attention to certain conditions and facts which to my mind are of the greatest clinical importance. I refer to the pathological anatomy of *incipient phthisis*, as that term is understood, or what is still oftener designated as *catarrh of the apex*. It was my intention to enlarge upon physical signs and their significance at this stage of lung trouble. But let it

suffice to say this: *if* we accept Buhl's observations of the disappearance of the products of croupous and catarrhal pneumonia, we are forced to this conclusion, viz.: WITH THE RARE EXCEPTION OF CASES OF ACUTE MILIARY TUBERCLE, NEARLY ALL CASES OF CATARRH OF THE APEX, IN PERSONS PREVIOUSLY HEALTHY, ARE CASES OF SUBACUTE GENUINE DESQUAMATIVE PNEUMONIA.

I say "if" advisedly, as in a future paper on phthisis itself the summing up of other authorities against Buhl is very strong. The bulk of evidence would show that his desquamative pneumonia is not entitled to be classed as a new picture of disease, but as the result of combination of the other pathological changes.

Still, as above stated, if the products of catarrhal and croupous pneumonia are always disposed of, we must allow that catarrh of the apex fits into the frame of desquamative pneumonia.

It will be remembered that chronic fatty degeneration is merely a prolonged desquamative process. The same process can scarcely exist in a subacute form. While elimination takes place, there is *regeneration* at the same time; and this fact is one of the chief factors in my hope of the successful treatment of consumption. Again, the râles heard in catarrh of the apex are fine crepitant ones, pointing to the alveoli as their seat; and secondly, the sputa are chiefly the same as in genuine desquamative pneumonia, large quantities of *cast-off alveolar epithelium* being the principal ingredient.

As before stated, I will, in my final summing up, once more recur to this subject.

INTERSTITIAL PNEUMONIA.

The healthy lung contains but little connective tissue, which, together with numerous elastic fibres, contributes to the formation of the lung-cells. This connective tissue also serves to connect the different lobules with each other, as well as to accompany the walls of vessels and of the bronchi.

In a comparatively large number of cases of lung trouble, we find, in the place of these traces of connective tissue, extensive sections of the lung converted into a tough, knotty, fibrous tissue. This is the result of chronic interstitial pneumonia.

Whether or not interstitial pneumonia ever occurs as a primary disease, is a mooted question. This would at first glance seem strange; but the fact is, that here we are dealing with a question of *time*. It is difficult, nay, almost impossible, to ascertain the original condition of lung trouble in a patient afflicted with the chronic interstitial form. But when you consider that there exists such a thing as an intensely acute interlobular pneumonia, it is not difficult to imagine cases primarily running a subacute course. Niemeyer, Nothnagel and others state that it "rarely ever occurs as an independent disease," and further that this is only the case where the long-continued inhalation of coal-dust and iron particles is the exciting factor. Even then, it is said, the cirrhotic condition is not primary, but the result of bronchitis.

Our subject is to be considered under two heads: 1*st*, *as an acute process, running a rapid course, and leading to the formation of pus;* 2*d*, *as a chronic proliferation and contraction of connective tissue, the result of a variety of processes.*

It will doubtless astonish you to learn that there exists such a thing as a separate and anatomically well-defined acute inflammation of connective tissue of the lung. If there is anything found in regard to it in medical literature, it is only hinted at as a possibility; this is owing to the well-nigh impossibility of clinically distinguishing it from other inflammations. In other words, a differential diagnosis is rendered impracticable by the sameness of the symptoms. The chill, the fever, the râles, and

the locality of the process are not pathognomonic ; nay, not even the sputa would necessarily settle the question. Still a close study of the history of a case which might be luckily seen in its inception, the inflammatory character of the temperature, and above all the subsequent course, differing in regard to time from that of croupous pneumonia, ought to elevate *acute* interstitial pneumonia to the dignity of an independent and separate picture of disease.

From the sparse material on this subject, we gather that this form of lung trouble occurs only as a consecutive one, by absorption of septic material. Its existence presupposes a centre for pyæmic infection. This centre may be only a cheesy mass. We do not know what are the immediate changes which would cause a cheesy mass to undergo suppuration. We do know that such changes are most likely to occur in broken-down constitutions (alcoholism), when the absorbed purulent matter would rapidly follow the course of the lymphatic vessels, which spread out in the interlobular and subpleural tissue. Again, the original cheesy centre may escape detection by its small size, though I am satisfied that its absorption is never total. But if, on the one hand, the original centre is not always demonstrable, and, on the other, the likelihood that a phlegmonous inflammation may as readily exist primarily in the lung as any other tissue, the question may be looked upon as an open one.

The chief anatomical characteristics of suppurative interstitial pneumonia are: turbid, dirty yellowish swelling of the connective tissue forming the support for the lung, infiltration of pus, that is, copious exit of white blood-corpuscles or hindered progress of lymph-corpuscles, and rapid breaking-down of the same. I beg you to remember that this process takes place in the interstices of the lung-lobules themselves, and not in the bronchial walls.

This disease occurs most frequently in newly-born children, whose pyæmic infection is then traceable to the puerperal infection of the mother. It may, however, also occur in adult life, in case of death by pyæmia. Indeed, we may say that all pyæmic infections involving the pleura and the adjacent connective tissue of the parenchyma, which are not caused by an embolic infarct, are *lymphatic interstitial pneumonia.* Or, to state it still more clearly: the embolus plays its part in the pulmonary

artery and its branches, spread out upon the lung-tissue proper, and the results consequent upon its presence are seen in the same tissue. The pyæmic infection is carried on *by the blood-vessels.* Infection from a cheesy centre (oftenest a lymphatic gland) is carried on *by the lymphatics*, which play their part in the tissue to which they are distributed—in our case the connective-tissue support of the lung. Hence, I repeat that, both for fact and simplicity, this process should properly be denominated *lymphatic interstitial pneumonia.*

The termination is probably always in death by suppuration and its consequences, which, as is known, is the most likely course in loose connective tissue (phlegmonous processes).

In passing to the second form of interstitial pneumonia, chronic proliferation and contraction of connective tissue, we at once find ourselves face to face with decidedly different views of different authors. One theory is supported by a single authority (Buhl), while all others (Rindfleisch, Ruehle, Stokes, Virchow, etc.) substantially agree. Buhl's position may be stated in a few words. The following are his claims, verbatim: "The name of chronic interstitial pneumonia signifies nothing else but the termination of genuine desquamative pneumonia." Having disposed of chronic fatty degeneration as one termination, he makes chronic interstitial pneumonia another, by the following reasoning: in consecutive desquamative pneumonia we have parenchymatous œdema, due to serous transudation only; in genuine desquamative a more plastic exudation, characterized by proliferation of alveolar epithelium and connective-tissue corpuscles. Where genuine desquamative pneumonia terminates in the cirrhotic condition, proliferation of connective-tissue cells predominates over the more superficial epithelial changes. The beginning of this proliferation takes place, in the acute stage, by the formation of spindle shaped and stellated cells (see last article), the first indication of embryonic hyperplasia of connective tissue. Cirrhosis of the lung proper, however, can only be spoken of when connective-tissue proliferation is enormous and appears as a fibrous cicatrix, in which the alveolar parenchyma and the finest bronchi are enclosed, obliterated, and have disappeared.

In order to simplify the question, I will add that, except by a few personal adherents, Buhl's proposition has been abandoned.

Other authorities, including such pathological histologists as Colberg, Rindfleisch, and Nothnagel, teach that chronic interstitial pneumonia is a termination of all forms of lung inflammations. At the same time, the existence of desquamative pneumonia is denied, of which Rindfleisch recently said that no one had ever diagnosticated a case but Buhl himself.

The objections to Buhl's desquamative pneumonia may be summed up thus: other observers have found and described in detail the same pathological changes, speaking of desquamation of epithelium of the alveoli. But they hold that this process is part of every acute lung or bronchial trouble, and not one which occurs as an independent disease.

In chronic interstitial pneumonia the inter-alveolar and inter-lobular connective tissue become the seat of inflammatory, nutritive changes, consisting of hyperplasia of the connective tissue, thereby causing an increase of the supporting structure at the expense of the tissues containing air. In the further progress of its proliferation, the newly-formed connective tissue undergoes the same changes as the connective tissue formed by inflammatory hyperplasia. At first soft and vascular, it gradually undergoes a process of *contraction*, converting it into a hard, bloodless tissue, occupying far less space than did the healthy parenchyma.

On section, the lung is found to be of one-half to one-third of its original normal size. It is firm, even to the consistency of cartilage, devoid of blood-vessels, and anæmic. Instead of the white color of connective tissue, we have a gray, slate color, due to pigmentation, for which the lung seems to have a predisposition, and more especially so in chronic interstitial pneumonia. Rindfleisch calls this "slate-colored induration."

In the midst of this hardened tissue, and throughout various parts of it, we find dilated bronchi and sacculated caverns, the latter an exaggerated dilatation. Excessive pigmentation is also characteristic of desquamative pneumonia; and hence Buhl dwells upon this as one of his reasons for making cirrhosis a termination of this disease only.

The next point to claim our attention is the important rôle played by the *pleura*. An autopsy of diffused cirrhosis of the lung always shows the pleura to be much involved. The two layers are adherent and transformed into a hard, thick, fibrous

sward, often one centimetre in thickness. Through this the lung is attached to the inner wall of the thorax. The thickest, densest portion is mostly at the apex of the lung, while farther down the adhesions get to look more like a cobweb.

By far the greater number of cases of cirrhosis of the lung have their origin in pleurisy, whether uncomplicated or as a concomitant of other acute processes. Corrigan (whose investigations justify the oft-applied name of *Corrigan's cirrhosis of the lung*) has shown that the process of chronic proliferation of connective tissue passes from the pleura to the lungs. This raises the question, whether all pleuritic processes (pleuritis sicca, serous effusion, empyema) lead to cirrhosis. In pleuritis sicca it is less likely, on account of the usually rapid absorption of fibrine.

There can be no doubt that cases in which absorption of the exudation has required a considerable length of time, lead to cirrhosis, as they likewise do to *retrécissement de la poitrine.* Laënnec believed that hemorrhagic pleuritis was most apt to lead to retraction. The true solution seems to me to be this: ANY PLEURISY (WHETHER SICCA, WITH SEROUS EFFUSION, HEMORRHAGIC, EMPYEMA, OR IN CONNECTION WITH CROUPOUS PNEUMONIA) MAY EITHER PRIMARILY PASS OVER INTO THE LUNG BY MEANS OF THE SUBPLEURAL CONNECTIVE TISSUE, AND CONTINUE AS SUBACUTE INTERSTITIAL PNEUMONIA; OR CIRRHOSIS OF THE LUNG MAY BE PRIMARILY CAUSED BY RETRÉCISSEMENT DE LA POITRINE, FOLLOWING THE ABSORPTION OF A PLEURITIC EXUDATION.

That is, primarily, only so far as the lung itself is concerned, since it presupposes a pleuritic trouble. This is the only form in which chronic interstitial pneumonia can lay a claim to being an independent disease. It is evident that the claim is but a slight one. Let me add that in croupous pneumonia it is not so likely to occur, on account of the usual restitution of the pleura together with the lung exudation.

Besides pleuritis, other primary diseases of the lung may lead to cirrhosis. Bear in mind, however, that the inflammatory products (exudations, secretions) do not themselves undergo such change, but are subject to cheesy degeneration. You may imagine the process as one which, in consequence of the pulmonary affection, manifests itself as an extensive interstitial pneumonia, starting from the original seat of trouble. The

question as to whether cirrhosis will supervene in any given case, is altogether a question of time, the same as in pleurisy. An exudation which does not begin to be absorbed directly upon the subsidence of the active process, or is not absorbed at all, acts as sufficient irritation of a subacute character. *Now, to any subacute irritation sufficient to give rise to renewed inflammatory exudation, the tissues respond by a development of connective tissue.* Chronic interstitial pneumonia, then, may occur as a sequel to or in connection with:

1st. *Croupous pneumonia*, for which we have the authority of Stokes, who quotes a number of cases uncomplicated by pleuritis, where, after the absorption of the croupous exudation, the cirrhotic condition existed and continued. The co-existence of a subacute pleurisy might in these cases have readily escaped the best observer.

2d. As a result of *catarrhal pneumonia*, Bartels found, in a number of cases which had run a chronic course, the lower lobes in a cirrhotic condition.

3d. As a result of *collapse of the lung*, either primarily or as a sequel to catarrhal pneumonia consequent upon the collapse.

4th. Consequent upon *gangrene and abscess of the lung.* You know that in the healing process the destroyed part is bordered by connective tissue. From this cicatrix the process of shrinking may extend to the rest of the lung.

5th. Interstitial pneumonia is said to occur as a complication of *chronic bronchitis*, the discussion of which we leave for the chapter on peribronchitis.

6th. The development of *cancer*, the presence of *hemorrhagic infarct*, and *apoplexy of the lung* lead to incapsulation by connective tissue, a sort of local cirrhosis. It is doubtful whether a whole lung can become involved in the process in such cases. In embolism (embolic pneumonia, so called) the pleura is secondarily involved. This is to be accounted for thus: the embolus originates in the right side of the heart or is carried to it by the peripheral circulation, and entering the pulmonary artery clogs up sections of lung circulation; but this is speedily made up by other branches of the pulmonary, whose compensating distention is assisted by the elastic yellow fibrous tissue. Hyrtl has lately established the fact that the branches of the pulmonary artery pass directly into the pleura. The pleural vessels are not in intimate relation with the bronchial arteries.

In a certain sense the deposit of tubercle, and more particularly the softening of tubercular masses, gives rise to interstitial pneumonia, "with nutritive exudation." (Virchow.) But this process is decidedly a local one, not extensive enough to be dignified by the designation of interstitial pneumonia. Through it originate the connective-tissue capsules, which serve to separate tubercular products from normal lung-tissue. When we come to speak of tuberculosis, I will endeavor to show that tubercular invasion is apt to supervene during the progress of chronic interstitial pneumonia, and often causes rapid death.

These articles do not concern themselves with diagnosis. Yet it may not be amiss to speak of the importance of the patient's previous history of the trouble; its inception; of the emphysema accompanying its further progress, and the consequent displacement of the liver, spleen, diaphragm, and heart, as well as to mention bronchiectatic caverns, which are often dry.

Two diagnostic points are of great value in this connection. They are: 1st, Almost total absence of fever during the progress of chronic interstitial pneumonia and until tubercular infiltration supervenes; and 2d, The character of the cough and sputa, of which Niemeyer says: "Hard attacks of cough, repeating themselves at long intervals, by the force of which large quantities of stinking sputa are expectorated, are pathognomonic of bronchiectatic caverns."

CASEOUS PNEUMONIA.

Having now considered the primary affections of the lung, acute and chronic, with their terminations, we pass to that condition, which even more than cirrhosis is the result, the frequent fate of their products. While in the event of resolution of the inflammatory product in pneumonia this product undergoes fatty degeneration, liquefies, and is absorbed, fatty metamorphosis may, on the other hand, be incomplete, the infiltration dry up, the cellular elements becoming atrophied and shrinking from loss of their watery contents.

There is no question that "EVERY FORM OF PNEUMONIA MAY, UNDER GIVEN CONDITIONS, TERMINATE IN CASEOUS PNEUMONIA; BUT IN NO FORM OF PNEUMONIA IS CASEOUS METAMORPHOSIS THE CONSTANT AND ONLY TERMINATION."

This doctrine naturally suggests the question: which forms of pneumonia are the most liable to terminate in cheesy pneumonia?

We find that for the picture of pathological conditions, which Buhl calls desquamative pneumonia, he claims the parentage of each and every caseous pneumonia, to the total exclusion of croupous and catarrhal pneumonia. I will tersely state his reasons therefor: cheesy degeneration is fatty degeneration, distinguished from the fatty metamorphosis by absolute anæmia and constant absorption of water, begetting paleness and dryness; while in fatty degeneration the parts are even more than normally moistened by constant flow of blood. The conditions for cheesy degeneration are found in desquamative pneumonia. Cheesy degeneration supervenes where a part supplied by a capillary trunk is suddenly cut off from its source of circulation, causing *anæmic necrosis*. It is this necrotic tissue which becomes caseous. Thus we have in cheesy degeneration various stages of pathological changes, an acute as well as a chronic one.

In the acute stage the lung presents the following appearances: it is augmented in volume as well as weight; the cut surface is of the color of porphyry red, displaying upon its face yellowish-white granules of various sizes, isolated or congregated.

The red color is due to hyperæmia. The lung does not contain much air. From the cut surface a large quantity of transparent, jelly-like fluid can be scraped off, in which are found, besides red blood-corpuscles, bullet-shaped or flattened, sharply-defined transparent cells, having one or more nuclei. These are the gelatinous, desquamating, and self-regenerating epithelia of the alveoli.

The yellowish-white deposits are dry, friable parts of tissue, void of air and blood. Their various sizes correspond to single alveoli, or more frequently whole alveolar spaces, or even a convolution of several. Microscopically they consist of *shrunken alveolar endothelium and necrotic connective tissue.*

According to Buhl, the red groundwork is the same as in genuine desquamative pneumonia, and the yellowish deposits merely anæmically necrotic parts.

It will not be amiss here to quote Niemeyer, in order to show that, as regards the pathological anatomy of what is called cheesy pneumonia, there is no great diversity of opinion. He says: "A PNEUMONIA WILL LEAD THE SOONER TO CONSUMPTION, THE MORE ABUNDANT IS THE AGGREGATION OF CELLULAR ELEMENTS IN THE ALVEOLI AND THE LONGER THE INFLAMMATION LINGERS, AS THESE TWO FACTORS TEND TO ENCOURAGE CHEESY DEGENERATION OF THE INFLAMMATORY INFILTRATION."

Passing to the pathological anatomy of cheesy degeneration in its subacute and chronic state, we find several possibilities of final termination of the acute stage.

One termination is restitution to the normal condition. This is accomplished by transitory fatty degeneration of the hyperæmic (red) groundwork, while the yellowish-white anæmic necroses shrink more and more by the continued absorption of their watery contents, that is, they become cheesy. This termination is likely to occur where the yellow masses are not very numerous, and at a tolerable distance from each other. Even then the lung contains cheesy tissue in various parts, always closely surrounding a minute bronchus.

The second termination is one likely to occur when cell-proliferation has not been so copious as to seriously compress the alveolar walls, and thereby cut off the circulation of those parts. The cheesy mass then becomes more and more inspissated, and the shrunken atrophic cells remain as mere detritus. The

organic substances gradually disappear, while calcareous deposit takes place, leaving at length a chalky concrement.

It is during these changes that the important act of *copious proliferation of connective tissue* takes place, as described in my letter on cirrhosis.

The chalky concrements are thus incapsulated, and then may undergo slow fatty degeneration. The spaces, out of which the fattily degenerated cells have vanished by liquefaction and absorption, are filled by connective tissue. Here, then, as elsewhere, *the caseous mass is the remnant of the original inflammatory infiltration and the newly-formed connective tissue its capsule.*

It is well to remember that all these conditions may be found in the same lung at the same time, to wit: normal integrity, fatty degeneration, and cirrhosis. This is due to the uneven distribution of the cell-infiltration, giving rise here to anæmic necrosis, there to cirrhotic induration alone, and again to fatty degeneration.

Remember that I stated, in my letter on desquamative pneumonia, that all authors substantially agreed as to the pathological finding in inflammations of the lung accompanying infectious diseases, the name alone not being universally recognized.

The question is merely, whether or not these appearances present such a well-defined, distinct picture of disease as to raise it to the dignity of a separate designation. Once more touching upon this subject, we may sum up the negation to Buhl's dictum as follows: besides the processes which Buhl calls consecutive desquamative pneumonia, other forms of inflammation occur in connection with acute infectious diseases, from simple hypostatic hyperæmia to lobular and lobar croupous pneumonia, which latter are not found at the most dependent portion of the lung, and not dependent upon hypostasis, and are said to occur during the height of the infection. This too can be explained. Careful observation in dissections will show the characteristic changes of organs in infectious diseases (for instance, the infiltration of Peyer's patches) to have come to a stand-still at the time when croupous or catarrhal pneumonia commenced. I think we are safe in setting up this doctrine: *inflammations of the lungs accompanying infectious diseases are of the hypostatic hyperæmic or œdematous kind* (*consecutive desquamative pneumonia*). *Croupous and catarrhal pneumonia and cirrhotic processes occur*

in debilitated persons after the infection has run its course; where they begin before the legitimate termination, the pathological changes characteristic of the original infection will be found to have reached their acme.

Applying this doctrine to the residuary products of a lung trouble accompanying infectious diseases, which products are all the more apt to undergo cheesy degeneration, because of the debilitated condition of the convalescent patient, we are safe in pronouncing: if (according to Buhl) *all* inflammations accompanying infections diseases, at any stage, were desquamative, then the subsequent cheesy degeneration cannot have been a croupons or catarrhal product. But since croupons and catarrhal pneumonia and cirrhotic processes occur during the disease as well as after, their products are present and subject to cheesy degeneration.

That products of inflammations accompanying infections diseases should be eminently prone to cheesy degeneration is not astonishing, when we consider that it requires an infection of no mean intensity to give rise to an accompanying pneumonia. The intensity of the invasion leaves the convalescing patient in a very debilitated condition.

From this we are justified in making this deduction: INFLAMMATORY PRODUCTS OF LUNG TROUBLE IN CONNECTION WITH INFECTIOUS DISEASES WILL TEND TO CHEESY DEGENERATION IN CASES WHERE CONVALESCENCE IS SLOW AND THE EXUDATION LINGERS FOR MORE THAN A VERY LIMITED PERIOD.

Next in frequency in the tendency of its products to cheesify, is true *croupous pneumonia.* Clinically it is so often impossible to distinguish croupons from "desquamative" pneumonia, that we are not able to establish from experience, at the bedside, whether croupons exudation, as such, often undergoes cheesy degeneration. Juergensen hints at the possibility of a desquamative process following croupons pneumonia (if we place it among infectious diseases) and leading to contraction, formation of cavities, etc. According to Ruehle, cheesy degeneration is most apt to supervene, where the upper lobe has been involved, since it has been shown that in such cases the disease drags on for a longer period than is common in the lower lobes.

The products of *catarrhal pneumonia* next claim our attention. In all cases of exquisite catarrhal pneumonia the same

sequel occurs as mentioned in connection with croupous pneumonia. The seat of catarrhal pneumonia is always in the lower lobes, less accessible to atmospheric influences. Besides, as you will remember, I have stated that both Bartels and Ziemssen have found only two cases, each of well-proven cheesy degeneration of products of catarrhal pneumonia.

In regard to *bronchial catarrhs*, *pleurisy*, and *hemorrhages*, let me tersely state that there is now, as there has ever been, great diversity of opinion regarding the caseation of their products, as well as of their leading more or less directly to consumption. The difficulty here lies in the well-nigh impossibility of positive clinical proof, of which I have spoken before. No doubt, you have often asked yourself the question: *does bronchitis lead directly to consumption?* Let me give you a few points in reference thereto: 1st. Catarrhal pneumonia (capillary bronchitis) does so rarely, and yet the bronchioles are nearest the parenchyma. 2d. Where it does occur, it is of the form which is consecutive upon general diseases and where the patient is much reduced. 3d. Bronchitis of specific tendency, born of scrofulosis, alone paves the way to caseation and consumption directly. Of this more in a subseqent letter on the relation of scrofula to consumption. The products of *pleurisy*, when slow to be absorbed and lingering, are the indirect cause of cheesy degeneration of such products in the lung tissue, which coexisted with the pleuritic ones. Remnants of pus in the pleural cavity, especially when incapsulated, may dry to the consistency of mortar, and thence become cheesy centres. I refer you to the letter on interstitial pneumonia, and repeat, what has been stated once before: *the inflammatory products existing at the termination of a lung trouble are subject to caseous degeneration. Interstitial pneumonia* (*cirrhosis*) *may begin at any time after the acme has been reached, and then tends to enclose, to encapsulate these products by proliferation and subsequent contraction.*

Hemorrhages, hæmoptysis, as well as bronchial hemorrhages, from any cause, are by no means only evidences of progressive phthisis. On the contrary, hemorrhage from any fulness of blood-vessels, by stagnation, may furnish the material for cheesy degeneration. Indeed, the rapidity with which this occurs is second only to its rapid decomposition by contact with atmospheric air and expectoration or its partial reabsorption, and may be

looked for when lingering for more than a very limited period. You are aware that caseous products were formerly called yellow tubercle. Niemeyer, following the teachings of Virchow, first drew attention to their being products of various forms of pneumonia. Still he held, and in this is supported by more recent authorities, that tubercles proper are present in these cheesy masses. In a future letter, on tuberculosis, I will endeavor to demonstrate that *both in primary chronic interstitial pneumonia, as well as in cheesy degenerated products of pneumonia, miliary tubercle may supervene.*

Other important points, touching upon the ultimate fate of cheesy pneumonia and its relation to consumption, acute tuberculosis, and scrofulosis, will be treated of in future letters.

A fourth termination, and one which may supervene during the active stage of pneumonic processes, is that of the formation of caverns, cavities. Great intensity of the inflammatory process and rapid degeneration (passing, as it were, over the acute stage of cheesy transformation) may lead by demarcation to the detachment of a slough. This process depends upon an augmented cell proliferation, surrounding and cutting off the capillary arteries; or it may be caused by embolism of minute arteries, a pyæmic infarct.

Caverns in close proximity may burst into one another, forming extensive caverns. Superficial ones may perforate the pleura, and produce pneumothorax.

Demarcating suppuration, the gradually developing granulations with continued suppuration (the pyogenic membrane), are factors too well known to dwell upon. Suffice it to say, the granulations may become a fibrous wall, with subsequent slight contraction of the original connective tissue zone.

These same granulations, however, may continue to furnish pus, which, decomposed by contact with atmospheric air, furnishes a fresh incentive for sloughing. By sloughing, layer upon layer of the cirrhotic wall is detached, the cavity enlarged, and previously dilated blood-vessels opened, causing a hemorrhage.

It is for these reasons that cavities, not exceeding the size of a pea, are the only ones where we may hope for a permanent healing process, by a *contracting zone of connective tissue, and the subsequent slow fatty degeneration or calcification (chalky transformation) of the limited contents.* And this favorable result

even is largely dependent upon the *number* of such small cavities and their *proximity* to each other, since, where small cavities are numerous, the breaking-down process may supervene before time sufficient has elapsed for the slowly progressing indurative processes; while their close proximity is a factor in favor of enlargement, by breaking down of walls and the union of one or more small cavities. Another method of enlargement is the junction of a cavity and a bronchus. In fully-formed cavities, in which cheesy masses develop slowly, the pleuræ have time to form adhesions. If rapidly, air may escape into the pleural cavity, and lead to pneumothorax and suppurative pleuritis.

Where shrinkage of connective tissue takes place to any great extent, *blood-vessels will be formed*, chiefly in connection with the pleura, a fact to which reference has been made before. These *newly-formed vessels do not penetrate deeply into the infiltration, but surround and supply it with constant though scanty nourishment.* This is similar to the relation between perichondrium and cartilage. The dense connective tissue surrounding old lesions is continuous on the outside with the thickened pleura, inside with the peribronchial connective tissue.

Large cheesy masses, therefore, are held in their periphery by cirrhotic contraction outside; they are moistened continually by the newly-formed circulation above mentioned, and thus the fibrous capsule encloses a chronic cavern, whose chances of enlargement are second only to those of a newly-formed cavity.

PERIBRONCHITIS.

No treatise on consumption, the conditions leading thereto, and the possible complications of these conditions, can be complete without a full understanding of such processes which, mostly as secondary consecutive forms of inflammation, play an important part.

Certain forms of peribronchitis are primary, independent, totally unmixed diseases from the beginning. Now, you will want to know why they are not enumerated as such in text-books, and a clinical picture drawn of them for our guidance. Distinct symptoms, capable of being detected by physical exploration, do not exist to such an extent or sufficiently different from other forms of chest trouble that a clinical picture can be made out, even with the assistance of an exact history of the case. As a rule, it may also be said that the subjective symptoms often escape the patient, until secondary destructive processes cause distinct discomfort, when we are then dealing with a tuberculous process leading directly to consumption.

Thus it is that pathological anatomy alone can establish the existence of such diseases as peribronchitis, some forms of interstitial pneumonia, etc.; and it is from pathological anatomists, like Rindfleisch, that we derive our facts. But no sooner have these pathological changes, as found in the cadaver, been properly sifted and described, than we find a number of the same observers aver that these same findings compelled them to fall back upon the old theory of the identity of all phthisical processes with tubercular degeneration. Thus Rindfleisch, who in his former work (pp. 334 and 345) describes purulent peribronchitis as a distinct form of disease, now says: "I feel it incumbent upon myself, on the strength of new observations, to widen the at present much contracted sphere of tuberculosis of the lung, and give support to the views of those observers who have never ceased to look upon consumption as essentially a tuberculous affection." Of course all proof in this connection properly belongs to a chapter on tuberculosis; and it is to this I must once more refer you, in order to avoid tedious repetition.

I will here confine myself to the description of peribronchitis, as spoken of by authors, on the basis of pathological anatomy, and again warn you not to attach too much clinical importance to the same.

Peribronchitis is an inflammation of the bronchial tubes, having its principal seat in the *adventitia of the bronchi and bronchioli*, and involving only secondarily the quality and quantity of the secretions of the bronchial mucous membrane. It is altogether an inflammation of connective tissue, all other processes following in its train. In this it resembles interstitial pneumonia, and, like it, presents two possibilities: peribronchitis with and without the formation of pus.

As often indicated before, let me repeat here that, as in all inflammations of the same family, these two conditions may coexist.

It seems natural to begin with the simple form, in which no pus is generated. *Simple peribronchitis* is always a chronic inflammation. This is all the more easily understood when you consider that an acute inflammatory process of connective tissue in any part tends to rapid transfusion (œdema) and equally rapid migration of white blood-corpuscles (purulent infiltration). A less rapid process, on the other hand, will tend to the formation of new tissue, rather than to the production of serum, mucus, or pus.

The simplest presentation of peribronchitis is where, in pneumonic processes, white fibrous layers accompany the finer bronchi and bronchioles without necessarily obliterating them. Such a process may be termed "diffused cirrhosis of the lung," where obliteration of alveoli and bronchi are accompanied by extensive substitution of condensed connective tissue.

Where, on the other hand, *multiple black fibrous knots* are found in otherwise healthy lung tissue, we may consider the process as one of *lobular cirrhosis of the lung*. We need only to remember that connective tissue is here a continuous tissue of the same kind, not varying, as that of the inner aspect of the bronchi and alveoli. So that in all these peribronchitic processes we are only dealing with a change in quantity, in which either the interalveolar or the peribronchial connective tissue may be predominantly hypertrophied.

You will remember that, speaking of the higher grades of

desquamative pneumonia, mention was made of the cellular hyperplasia of endothelium of the arterial coats. This same condition supervenes in peribronchitic processes. A bronchial tube becomes uneven, knottily thickened, and fibrous, with degenerating endothelial cells.

This is one of the most frequent chronic disturbances, and is very properly termed *peribronchitis nodosa.* Peribronchitis nodosa may occur independently; indeed, the same individual may several times during lifetime suffer from localized processes of this kind without active symptoms, just as slight pleuritic adhesions occur.

Mostly, peribronchitis fibrosa and nodosa occur together, and, by continued shrinkage of the hypertrophic tissue, may lead to atrophic ectasia of alveoli and bronchioles as well as dilatation of the blood-vessels contained therein.

In very intense, acute cases of course the tense traction, by shrinkage, upon the arterial branches and capillaries, may lead to complete interruption of the circulation, and the natural local consequences, anæmic necrosis or gangrenous throwing off of a great portion of a bronchus. It more frequently occurs, however, that the relatively small section of necrosed tissue remains in organic contact and undergoes cheesy degeneration.

When local gangrene occurs in this way, it is of course an acute or subacute process, but as such strictly localized and not diffused, while the universal cheesy degeneration is altogether a chronic process. Like diffused cheesy pneumonias, these cheesy lobular masses are encapsulated by black cirrhotic tissue, and, when quite small, may be reabsorbed. Such multiple lobular pneumonias have been classified under the head of chronic tuberculosis, they being looked upon as a slow cheesy metamorphosis of existing tubercle. In reality they represent merely various stages of a connective tissue process, and the results are governed by the *length of time necessary for their development;* the acutest form gives rise to alveolar inflammation, the subacute moves towards the bronchioles, and the chronic *form* (whether as a remnant of an acute process or of slow progress from the beginning) lays hold of the coarser ramifications of the bronchi.

In conclusion I will add that a close analogy seems to exist between desquamative pneumonia and peribronchitis in this: as desquamative pneumonia is a localized expression of some general

disease (*i.e.*, being the form of pneumonia which occurs in connection with acute infectious diseases), so may peribronchitis, which plays its part in the same tissue, be a localized expression of general disease, possibly chronic.

Peribronchitis purulenta. It is my opinion that of all forms of respiratory affections, this one is the least understood. In vain do we search the works of even the more recent authors for a clear, well-defined picture of this disease. This is all the more astonishing, as purulent peribronchitis occurs as an independent, uncomplicated disorder. Its histological factors have been best described by Rindfleisch, and it is to his observations we will address ourselves.

Purulent peribronchitis is characterized by *acute purulent infiltration of the finer bronchi* (those wanting in cartilage, muscle, or glands), *involving their whole thickness, from the cellular coat to the mucous membrane.* It rapidly extends along the lobules and into the alveolar walls. In its train may follow the results of all rapid purulent infiltrations—loss of substance, abscesses, ulcers of the bronchi, vegetable parasites of a low order. This purulent softening must not be confounded with anæmic necrosis, though the dead mass may become cheesily degenerated, and, when of large extent, form cavities. In the formation of cavities a number of terminal bronchi, with lung lobules belonging to them, will be dissolved in the pus, or the purulent infiltration may proceed towards the bronchioles, and find its limit only where cartilaginous rings begin. As in simple peribronchitis, there appears to be (according to Buhl, of course) an analogy between desquamative pneumonia and purulent peribronchitis in this: that in desquamative pneumonia the parenchymatous swelling is of a proliferating, productive character, while in purulent peribronchitis it is the same process with a destructive tendency.

In consequence of the swelling and removal of bronchial tissues, the entrances to the alveoli are often well-nigh clogged. The alveoli are at this time full of air, and, expiration becoming impossible, an *acute multiple lobular emphysema* supervenes.

This is one of the few pathognostic symptoms of purulent peribronchitis, and may result in pneumothorax from several small forced ruptures at once.

In regard to diagnosis, which you know we do not strictly enter into in these letters, I cannot forbear to mention the fact that the chills and continued fever, as evidences of rapid formation of pus, are guiding-points for a differential diagnosis.

So far as known, purulent peribronchitis does not become chronic. This may be inferred from its character as an acute purulent infiltration. In case of any less rapid course (as, for instance, when successfully combated by treatment), the primary results may become circumscribed by *cheesy induration of the purulent infiltration* and such weak condensation of the surrounding connective tissue as to render constant repetitions almost certain.

In regard to purulent peribronchitis, the same rule holds good to which, in other conditions, I have so frequently drawn your attention, namely, that of *combining with other forms of inflammation*—in this instance with desquamative pneumonia and knotty peribronchitis—frequently forming the last stage of the series.

Niemeyer had a favorite aphorism, to the effect that "the greatest danger in chronic inflammatory processes of the lung is the supervention of tuberculosis." Buhl is, no doubt, in the right when he desires to have this modified so as to read, "the danger consists in the addition of purulent peribronchitis." And he seeks to establish the importance of the occurrence of purulent peribronchitis in any case by the following deductions: the formation of pus in this trouble is not to be found in the migration and infiltration of white blood-corpuscles, but is accomplished on the spot. Migratory cells do not reach the surface of unharmed mucous membranes.

The connective tissue corpuscles and endothelium of the lymphatics produce pus-corpuscles freely from their nuclei, rapidly degenerating themselves and leaving the pus-globules free. Though not strictly proven, it is now one of the theories under investigation that the formation of lymph and white blood-corpuscles in the healthy state takes place from the endothelium of the lymphatics, confined to certain parts of the organism, but that under pathological irritation they may be produced in any part where lymphatics or connective-tissue cells analogous to endothelium exist. And let me add here, that it seems plausible to me that length of time as well as severity of attack is an element,

and that the two are the chief factors in producing destruction in the shape of *rapidly formed endothelial and connective-tissue cells, on the spot*, in contradistinction to more chronic and less severe invasions, which give rise to slow processes in the shape of *migration of white blood-corpuscles*, etc., resulting in hyperplastic conditions.

TUBERCULOSIS.

Our present conceptions of the nature of tuberculosis are to a great extent based upon the investigations of Virchow. It was Virchow who first demonstrated that cheesy degeneration, far from being a specifically tuberculous process, supervened in the train of other neoplasms, cancer, sarcoma, etc., nay even quite frequently as a sequel of chronic inflammations; he taught us to become acquainted with the true character of lobular hepatizations in the lung as well as cheesy peribronchitis, which had been so often confounded with tubercle, and pronounced as the typical form of tubercle the miliary eruptions of serous membranes.

The definition given by Virchow of tubercle, aside from a few modifications, holds good to-day: "Tubercle is a neoplasm which appears in the shape of small nodules, always originating from connective-tissue strata, consisting of densely crowded small round cells. This neoplasm has a limited lease of life, very soon degeneration of its elements sets in, always beginning in the centre of the nodule; in most cases the change is into cheesy degeneration. Apart from this local malignity, the neoplasm possesses another trait, which places it in the category of malignant tumors: a well-marked tendency to generalization over the whole organism."

We may look upon these propositions of Virchow's as the foundation for the present views on tuberculosis and endeavor to amend them in accordance with newly discovered facts.

The next development was that Langhans drew attention to the almost constant occurrence of *giant-cells in the interior of tubercle.* Virchow, too, had spoken of the occasional presence of giant-cells, but Langhans was the first to announce them as a typical occurrence. He pointed to the peculiarly typical nature of tubercular giant-cells in contradistinction to the well-known giant-cells of sarcoma and to other neoplasms; he dwelt upon the peripheral location, the uniform size, the radial arrangement of their nuclei, and thereby characterized them as a growth *sui generis.* The protoplasm of these giant-cells is distinguished by uniform, rather dark nucleation, and in superficial observation

bears some resemblance to certain forms of bacteriæ. Some observers hastened at once to pronounce the dark granula of the giant-cells as micrococcus; the simple reaction with a potash solution, which at once dissolves the granules, when moderately warmed, dispelled this opinion.

Further pursuit of Langhans' discovery led Koester to find tubercle in a locality where up to date they had escaped all observation, namely, in the *granulations of fungous inflammations of joints.* Indeed, the occurrence of tubercle in this disease seems to be inexceptional, and their aggregation often visible to the naked eye.

Next Schueppel found the same giant-cells in *cheesy or apparently purely hyperplastic lymphatic glands.* Here they are only to be seen microscopically, not so much on account of their smaller size—they measure from one-third to one-half of a millimetre—as because they are not sufficiently distinguishable from the surrounding parenchyma of the lymphatic gland.

Again, according to the researches of Friedlander, a constant and prolific eruption of tubercle was found in *scrofulous abscesses and ulcers of the skin*, as well as in *caries.*

Thus we see that all of the most important scrofulous diseases are linked with the development of tubercle.

In addition to what has been said of the past history of tubercle, a rapid glance at its anatomico-histological character is necessary for a thorough comprehension of its subsequent fate.

Tubercle is a neoplasm of microscopical dimensions, of a diameter of $\frac{1}{12}$–$\frac{1}{6}'''$. Tubercle has within itself no blood-vessels, though surrounded by a network of blood-vessels, and itself is found in the connective tissue accompanying lymphatics and other vessels, especially the *adventitia of the more minute arteries.* Histologically, tubercle appears to be analogous with all lymphoid structures of the human body (Malpighian bodies of spleen, lenticular follicles of intestines).

Under the microscope tubercle is seen to be a bullet-shaped mass, commonly from two to ten of them closely aggregated together, so that the individual tubercle is really composed of a group of bullet-shaped masses. The real histological character of tubercle has been found by E. Wagner to consist of: *a network of most minute fibre* (reticulum) in which we find *cells which, both as to number and size, increase from the periphery to the centre.*

In regard to these cells it may be well to cite opinions as to their different character in connection with their location. Imagine, therefore, the above-mentioned body of delicate fibrous tissue with cells, of which there are three kinds:

1st. So-called *cystoid bodies, found chiefly in the periphery of the fibrous groundwork.* Several authors contend that these particular elements of tubercle are migrated lymph-corpuscles or white blood-corpuscles. This is gainsaid by the fact that tubercle are much smaller, as also are their nuclei smaller and more shining. Again, the centre of the fibrous groundwork are giant-cells, so that peripheral cells would have to increase in size, instead of undergoing the well-known fate of tubercular cells to the contrary.

At the end of the chapter on tuberculosis, I will again summarize the accepted theories of the present time. The analogy between tubercle-cells and lymph-cells is now under investigation. It may be reserved to histologists of the future to show the analogy between tubercle-cells, lymph-cells, and white blood- or pus-corpuscles; and while it is not requisite that tubercle-cells (any more than pus-corpuscles) should grow after migrating, it may yet be shown that their condition is *produced within the lymphatics and capillaries, or during their migration through the vascular walls, or both.*

The second form of tubercle-cells are the *giant-cells*, and these are situated *in the centre of the fibrous network.* The increase in size is so marked as they approach the centre, that the most central giant-cell is always superior in size to its surrounding neighbors.

Schueppel (1871) endeavored to invest the giant-cell with the dignity of a specific characteristic of tubercle, but as giant-cells exist on the inner surface of periosteum and elsewhere, this opinion does not hold good.

3d. Besides cystoid and giant-cells, we find cells of *epithelial character*, which as regards size and situation appear to be a transition between both (Buhl). The smallest giant-cells are the largest of this species. They fill the meshes of the reticulum surrounding the giant-cells; become smaller as they approach the peripheral cystoid cells, and appear to lose themselves into the cystoid cells on the one hand, as they do into the giant-cells on the other.

Where giant-cells do not exist, the epithelial cells may form the centre, or, in their absence, the cystoid cells have the ground to themselves, and are then not to be distinguished from lymph-follicles.

Two things seem to me possible in connection with the history of tubercle-cells. Since the peripheral cystoid cells are not supposed to be the juniors of the giant-cells, not a transition cell developing from the periphery to the centre; since tubercular ulceration and disintegration begins in the centre, as do all ordinary ulcerative conditions leading to destruction of tissue, it might be worth while to investigate whether the two following propositions are not founded on fact:

1st. THAT THE PROLIFERATION OF THE VARIOUS KINDS OF CELLS (CYSTOID, EPITHELIAL, AND GIANT) DOES NOT TAKE PLACE FROM THE PERIPHERY, EXCEPT PERHAPS IN THE BEGINNING, BUT FROM THE BOTTOM OF THE PREVIOUSLY DEVELOPED CONNECTIVE-TISSUE NETWORK. This would be analogous to the process of repair in varicose ulcers treated by cutting off the peripheral circulation, when reparative nourishment of the ulcer is accomplished through tissues underneath.

2d. That, as is the case in inflammatory conditions, the centre of the existing condition produces not only more fully developed cells (as shown in the prolific development of granulations in a wound), but this centre is invariably the starting-point of suppuration and disintegration.

As long as the tubercle granule is still growing, there is *increased activity of proliferation in the whole surrounding tissue*, which ends rather rapidly in fatty degeneration or calcification of the central giant-cells, and in hypertrophy of connective tissue from the cystoid cells of the periphery.

It is but natural that the origin of tubercle has always attracted a large share of attention, and every tissue, beginning with the circulation and ending with the cells of the immediate surrounding of tubercle, has been charged with its paternity. I will give you the present views about the paternity of tubercle in a few words:

Remember, as above stated, that tubercle essentially consists of giant-cells, next epithelial, then cystoid cells. Either of these three may be developed from connective-tissue corpuscles (according to Langhans), likewise from endothelial cells (according to Klebs, Rindfleisch, and others). Then Waldenburg, in his excel-

lent work on tuberculosis, endeavors to show the origin of tubercle in *capillary emboli caused by corpuscular elements.* For explanation of these theories, I have to refer you to the works of these respective observers.

The limit of my letters will only allow me to confine myself to tubercle in the lungs. In these, as in other parts, we find them only where connective tissue, lymphatics, and minute arteries with endothelial (lymphatic) coat are found. The part which these tissues play in the development of tubercle, is best described by Buhl: "Connective tissue, its interstices and vessels become animated to formation, wherever tubercle is located in the alveolar walls as well as the alveolar epithelium (as part of the continuous lymph-endothelium); likewise by the sub-pleural extension, the pleural endothelium; when tubercle is located in bronchial walls, the endothelium of lymphatics and epithelium of mucous membranes will take on prominent action. But all productive activity accompanying the formation of tubercle will only make itself felt so long as the tubercular growth is fresh and viable, and will cease and become retrogressive, as soon as the development of tubercle ceases and its degeneration begins."

Recent tubercle is soft as jelly, and has a grayish color. When it has reached full growth, which, from the fact of its being non-vascular, can never be considerable, fatty degeneration or horny calcification begins in the centre, and the growth looks yellowish and opalescent. This is characteristic of retrograde action on the part of tubercle; it becomes as a fibroid-kernel, and may be entirely reabsorbed.

In consequence of compression of the most minute arteries, the whole tract supplied by them becomes void of blood and undergoes *anæmic necrosis and rapid cheesy degeneration.* The whole of these together may sometimes form tuberculous caverns of small dimensions with cheesy contents, but here the action of tubercle proper ceases, since other processes originate from their neighboring tissues.

What has been taken for yellow tubercular masses, as spoken of in my introductory letter, is microscopically found to be a tubercle-lymphoma (tumor of that character, if you choose) with central degeneration; if large, as a necrotic group of alveoli, with their desquamated epithelium around and between them, ready to undergo cheesy degeneration.

Let us now pass from the pathology of tubercle to the consideration of the further course of acute tuberculosis. Miliary tubercle with the cheesily degenerated alveoli, lie either in healthy lung-tissue, or the lung-tissue will be undergoing fatty degeneration; in the apices, where tubercle and its masses are chiefly located, cirrhotic processes supervene, but not to any great extent. It is this which makes diagnosis ofttimes so difficult in the absence of decided physical signs.

These letters do not concern themselves with diagnosis, but I cannot refrain from impressing upon you the following: Where percussion and auscultation yield almost negative results; where the patient himself manifests no special subjective chest symptoms; where continued high temperature, not readily affected by quinine, a *rapid pulse* and *equally rapid emaciation* supervene, every careful physician should carefully inquire into the previous history of the patient. Nor should he neglect to search the fundus of the eye, the choroidea for freshly-developed tubercle. This *en passant.*

Ofttimes tubercle may in the general mass of cheesy degeneration have become obsolete and disappear, when it would be difficult to determine whether or not the mass before us is the result of slow peribronchitic processes or cheesy pneumonia, representing remnants of acute forms of lung trouble. The presence of a more recent crop of tubercle attests the character of the mass before us. At the same time it furnishes us with a hint as to the probability of a future permanent cure, which hope of course rests in the chances of reabsorption. Unfortunately, however, the next step in the development of tubercle is found in their movement into the bronchioles by local infection, and what in the alveoli was a closely condensed mass is found at greater intervals to correspond to the anatomical site of the minute bronchi.

I come now to speak of the etiology of miliary tubercle, and more particularly of the theory of its being a *specific infectious disease*, a disease of absorption of toxic matter. The literature on this subject is very extensive, and it will be my endeavor to summarize the evidence upon which this theory rests:

1st. MILIARY TUBERCLE IS FOUND AND DEVELOPED WHERE CHEESY DEGENERATIONS EXIST IN SOME PART OF THE BODY. This fact was known to Laennec and others, and accounts for his distinction into gray and yellow tubercle, yellow tubercle signify-

ing with him what are now known to be existing cheesy deposits.

From these centres particles are absorbed by the blood and lymph vessels, causing the multiple eruption of tubercle in one or several organs of the body, and nothing else. This, therefore, is self-infection pure and simple, resembling the invasion by other septic matter, as in septicæmia and pyæmia.

This proposition naturally gives rise to the question: Have cheesy parent deposits invariably been present, where tubercular eruption has taken place? This question is affirmed by some, negatived by others. As far as statistics of dissections are available, the result appears to be that in from eight to ten per cent. of deaths by miliary tuberculosis, no previous deposits were found at the autopsy.

The results of trials of inoculation are in accord with this. Where material was used which was not of inflammatory origin, the point of inoculation in each case first became inflamed and furnished products of inflammation, which afterwards became cheesy, and it was from this often rapidly formed cheesy centre that self-infecting absorption took place (Waldenburg).

2d. CHEESY DEPOSITS, LEADING TO MILIARY TUBERCLE, CAN BE TRACED BACK AS REMNANTS OF FINISHED INFLAMMATORY PROCESSES. Of course in order that absorption and infection may be accomplished, the caseous deposit must not be incapsulated, not surrounded on all sides by firm fibrous tissue.

3d. Another proof of the origin of tubercle by absorption is in *infection by contiguity*, probably due to absorption by local lymph-vessels. "Tubercle namely appear in greatest aggregation in the immediate neighborhood of the cheesy mass and multiply from thence in eccentric lines." The oldest tubercle are nearest the deposit, the youngest most remote.

Here we would almost have a basis for believing with Rindfleisch, in the existence of chronic tubercle, if we would and could distinguish as follows:

As acute tuberculosis, those processes which result from a rapid absorption into the general circulation of caseous material, producing constitutional symptoms (fever, etc.), and running a rapid course.

As chronic tuberculosis, those processes which are local from the beginning, allowing only of neighboring infection by means

of local lymphatic absorption and holding its own for a time, without falling a prey to absorption into the general circulation.

No such distinction, however, can properly be made, chiefly because localized tubercle is doomed to speedy death.

4th. Another proof of the specific character is the seat of miliary tubercle *in connective tissue well supplied with lymphatics*, and their analogy with the histological formation of the normal lymph organs of the body.

Having thus passed rapidly over what is known clinically of actual absorption, I draw your attention to what is claimed to be another mode of transfer of virus, viz., by *thrombosis* (Panum, Waldenburg, and others). Let me at once say, that thus far this is conjecture and has never yet been observed or demonstrated. But absorption and thrombosis are not necessarily irreconcilable. Buhl has endeavored to show that bacteriæ are the intermediate factors between a thrombosis and its partial absorption, by asserting that the corpuscles in the interior of giant-cells are in great part staff-bacteriæ.

I regret that it would again lead too far out of our prescribed path, were I to prove to you that bacteriæ are present in all necrotic breaking-down tissue, markedly so in the centre of ulcerative processes, just as in this case the giant-cells are the centre of tubercle and the first to undergo degeneration.

5th. Still another pillar for the support of the infection theory is the transfer as between husband and wife. As regards the proof of this, I will say that quite a number (I am sorry to say there are no positive statistics) of well-authenticated cases of transfer between man and wife have been recorded, similar to syphilitic infection.

6th. It is held by most authors that tuberculosis never goes hand in hand with other infections. This is true of carcinosis, typhoid, pyæmia, etc., but I am not sure whether it is equally true in regard to *malaria*, that bane of mankind, which according to several authorities does go intercurrent with tuberculosis. And I wish to impress upon you my oft-expressed belief, that *malarial infection is the immediate cause in every new country of breaking down and absorption of caseous products, and the occurrence of acute miliary tuberculosis.*

7th. Last but not least, the supporters of the infection theory look upon the success of the inoculation of true tubercle upon

animals, as an incontrovertible proof of the correctness of the absorption and infection theory of tuberculosis.

From all this evidence we are justified in speaking of general miliary tuberculosis as an acute infectious disease, in the sense that the own body furnishes the virus for the infection.

Let us now inquire in how far tubercle may be said to be a product of inflammation. I do not propose to engage in any discussion or examination of whether we may properly speak of a tuberculous inflammation, as this would amount to a war of words.

We have just seen that acute miliary tuberculosis is regarded as an acute infectious disease. Its character therefore is that of a general disease, and in order to be inflammatory in the true sense of the word, it would have to be strictly localized and produce local inflammatory products.

Therefore we may conclude to speak of *acute miliary tuberculosis as an inflammation, where simultaneously with the appearance of inflammation, tubercle-lymphoma are developed, which remain for a short time confined to the tissue then involved.*

Now you will ask: Is there a double manifestation of the tubercle-granule? Is there a secondary infectious miliary tuberculosis, taking its material from a cheesy centre, as well as a primary tuberculous eruption, which takes place during the existence of an inflammatory process, and at the site of that inflammation? In a certain sense, yes. According to Rindfleisch and others, the tubercular manifestations coexisting with inflammation occur in scrofulous individuals, and the products of such inflammation are marked by copiousness and a tendency to linger. Others again do not call products of that nature scrofulous, but dwell only upon the fact that they occur in individuals of feeble resisting power and general vulnerability. The supposition is in any case that before tubercle is formed, the products have undergone cheesy degeneration to some extent. Since Rindfleisch claims that the local as well as general manifestations are all based upon scrofula, I have thought it best to give his views unbroken in the subsequent chapter on "consumption."

So you may look upon tuberculosis as twofold in its manifestations.

1st. As a *local neoplasm*, at the site of subacute or chronic

inflammation, where the progress is confined to infection of the direct neighboring parts, and,

2d. As an *acute infectious disease* with all the attributes of infection, taking its origin by absorption from the localized centre.

It is to the first, to local tuberculosis, that I wish to invite your attention. Tubercle may occur in almost any part of the body, but it may be laid down as a rule that it has a certain predilection for serous membranes. Favorite sites are the pleura, pericardium, pia mater, peritoneum, the synovial membranes—in short, in those parts where lymphatic absorption is always active and the communication with lymphatic glands free and the glands themselves numerous. *Thus lying within a territory where changes are rapid, where transudation and exudation as well as absorption are very active, the ready general dissemination of tubercle from a localized centre is easily understood.*

The tendency to rapid distribution, governed by laws of which we know nothing, and their well-known tendency to equally rapid disintegration under certain conditions, justifies us in pronouncing tubercle capable of *great local malignity.* We find it so in lupus, in the chronic inflammation of joints known as synovitis fungosa, and what is of more interest to us just now, in the lungs. Here, often grouped together in a heap, they follow the course of the connective-tissue stratum of the bronchi. The intimate relations between this stratum and the subpleural connective tissue, invites the belief that the starting-point of tubercle in that locality is to be sought for in the pleura.

We find alongside of tubercle nodes always a more or less copious collection of small round cells, indicative of inflammatory irritation. The clinical picture of local tuberculosis is thus always one of a subacute or chronic inflammation. Thus three possibilities are presented:

1st. The tubercles and the inflammation are co-effects of the same cause.

2d. Tuberculosis calls forth a secondary inflammation.

3d. Tuberculosis is caused directly by the inflammatory process, consequently a secondary phenomenon.

You are already aware that this last theory is based upon the proofs furnished by Buhl, that in nearly all cases of general tuberculosis a cheesy parent-focus is found. And we have the equally well-known artificial creation of tubercle by creation of cheesy

products in animals. That in nearly all cases of death by general tuberculosis some cheesy focus should be found, is not at all astonishing. Few persons die, who have not carried within their body a cheesy product of greater or lesser dimensions during life. And after death from general tuberculosis, it is natural to suppose that amidst a quantity of tubercle, some of the older crop will be sure to have undergone calcareous metamorphosis. It is thus in a local sense that we may speak of "chronic tuberculosis."

We come now to dispose of Villemin's inoculation of tubercle in squirrels and the development of tubercle within their organs. The trouble about it is, that the *disease following the inoculation of animals is not tuberculosis.* What has been produced has neither the typical structure of tubercle, nor are any giant-cells present. The fact is that these *artificial nodules are lobular pneumonias*, which consist of the appearance of multiple centres of irritation with ready tendency to cheesy degeneration.

In disease caused by these cheesy masses, we are dealing with a chronic intoxication (chronic pyæmia) of animals, from which we have no right to make any deductions touching tuberculosis in man.

All that has been said above, holds good for the relations of the various inflammations of the lung to tubercle. Let me repeat:

Tubercle occurs, 1st. As a more or less independent *local manifestation* with local malignity, manifested by neighboring infection and rapid disintegration. Its favorite site are serous membranes. What agency primarily gives rise to the formation of tubercle, we do not know to-day any more than formerly. Nor do we know why it has a great tendency to the breaking down of its elements, but we conjecture that this occurs when a person's circulation is out of order (qualitative as in malarial poisoning, quantitative as in anæmia) or the equilibrium of the central nervous functions are disturbed. For those whose lymph organs are naturally vulnerable by inheritance, a mere trifling cause may suffice, usually a gastric or bronchial catarrh.

2d. As an *acute infectious disease*, taking its material for absorption from one of its own local deposits or an already existing cheesy deposit or chronic suppuration. General self-infection is the result. But let me impress it upon you, that before this general absorption takes place, tubercle must be formed on the site of caseation or suppuration. So that tubercle occurs,

3d. Locally again, inside of and in connection with the *cheesy remnants of finished inflammations*, and with *chronic suppuration*. Without further reference to other cheesy centres (scrofulous glands), or to chronic suppurations (chronic inflammation of joints, synovitis fungosa), we will address ourselves to remnants of inflammation of the lung and chronic suppurations of it and the pleura. In going over this ground, the repetition of some things which have been spoken of in previous chapters becomes unavoidable.

Of possible cheesy remnants of lung trouble we have: those of *croupous pneumonia*. This possibility is denied by Buhl, while all others allow it, but specify that it is a rare occurrence; either extreme—total and rapid restitution or equally rapid gray hepatization being the rule.

Those of *catarrhal pneumonia:* also denied by Buhl. Niemeyer claims for it frequent caseation. Others, more cautious, place it among the possibilities.

Those of *desquamative pneumonia:* to which form of inflammation alone Buhl credits the caseation of products. Rindfleisch and Juergensen allow for the pathological processes of this form, but not for the name. Others deny its existence, and call the inflammation accompanying general diseases catarrhal pneumonia.

In connection with *interstitial pneumonia:* in suppurative infiltration, as in gray hepatization and gangrene of the lung, the process almost invariably runs such a rapid course as to almost preclude the possibility of any other termination than death. We may imagine, however, that there exists such a thing as a subacute stage in which the formation of pus and of connective tissue go hand in hand, or, what is more likely, that a localized suppuration (if that ever takes place in such loose tissue) may be followed, and more or less encapsulated by newly-formed connective tissue. Here, then, we have the pus of a cold abscess drying up, thickening, and assuming a mortar-like consistence. This too is caseation. The occurrence of tubercle in newly-formed connective tissue, in cases where chronic interstitial pneumonia occurs as an independent disease of the apex, or in connection with the cheesy remnants of inflammations, may be imagined thus: as long as induration has not commenced, and the process is still one of proliferation, the ground is ripe for the

development of tubercle, for we have the authority of most observers to the effect that "tubercle of the lung locates itself only where connective tissue, minute arteries, and lymphatics are found."

Perhaps the most interesting connection, however, consists between the *products of pleuritis* and the formation of tubercle. We may safely exclude pleuritis sicca, with its fibrinous exudation, since there absorption is the rule, or at most limited adhesions. But in connection with the sero-fibrinous, serous, and purulent effusions, the proofs are manifold of an *etiological relation between acute miliary tuberculosis and the absorption of pleuritic exudations.* It has been found that after the somewhat rapid absorption of pleuritic effusions, tubercle has been developed on the spot as well as that general tuberculosis subsequently supervened.

The formation of tubercle in the pleura occurs as soon as the active symptoms have run their course and the exudation is perfect.

As stated in the beginning of this chapter, the favorite sites of tubercle are serous membranes. Now, of course, a crop of tubercle may have existed in the pleura, just previous to the lighting up of the inflammation, yes, may have been the local exciting cause.

Upon the completion of the exudative process, the pressure of the blood-vessels and lymphatics is very great, and the function of the absorbents greatly interfered with, interrupted, indeed, by their thorough soaking. Here, then, the lymphatics are completely clogged up, and the relation between waste and supply entirely suspended. Diuretics and other remedies are used, the lymphatics are partly freed, and rapid absorption takes place. Now, in this ready absorption into the open lymphatics enters also the tubercular virus, which in that respect has an equal chance with the exudation. It is from this point that general tuberculization begins.

But here we are at once confronted by another question: is the formation of tubercle in other organs due to rapid absorption of the pleuritic exudation? I do not think we are justified in assuming this. The injection of other foreign bodies, in short their introduction into the circulation direct or by hypodermic injection in animals, does not, as you now know, produce miliary

tubercle, but inflammatory foci, which are nothing more nor less than multiple lobular pneumonias. Now, a difference may exist between foreign corpuscular matter from without and the products of disease within the body, though the present accepted theory is, that while many cases of acute miliary tuberculosis originate from self-infection, it does not necessarily require a special specificity of infecting material, but retrogressive products of inflammation and detritus suffice, which may be formed by a variety of inflammatory processes. Temperature and other factors may play an important part here; but of this we do not know anything.

So that I think we may, from these premises, set up the following propositions:

1st. After the absorption of a pleuritic exudation, general tuberculosis frequently supervenes. This has been demonstrated by autopsies in cases of death in adults almost exclusively, the age of development being comparatively free, which may be readily accounted for by the rapid changes in early life.

2d. The source of tubercle in these instances is either tubercle or some other cheesy centre already existing in the pleura, or both. In addition to what has been said under that head above, we will look upon the probable results in case of empyema. Rapid absorption of pus in any quantity would give rise to pyæmia and early death, and not to tuberculosis. Let us imagine a purulent remnant encapsulated in the pleura and gradually becoming inspissated, drying up from still slowly progressing absorption, and undergoing cheesy degeneration. Now, whether a general tuberculosis would be lighted up at some future time, or whether a sudden recurrence of pleuritic symptoms would take place, we will in both cases be compelled to look upon the encapsulated remnant as the direct centre of absorption.

But even in cases where no traces of an older crop, of an original stock of tubercle, or cheesy mass can be found after death, we still have an explanation for the occurrence of general tuberculosis so soon after reabsorption.

The absorbed matter may pass on its way to the general circulation through lymph-glands, which are already in part cheesily degenerated, either from hereditary influences (scrofulous), or by more recent disease, and there readily take up the detritus. So we may formulate:

3d. A PLEURITIC EXUDATION, BECOMING ABSORBED AND PASSING ON ITS WAY THROUGH LYMPHATIC GLANDS ALREADY CHEESILY DEGENERATED, WILL START ACUTE MILIARY TUBERCULOSIS FROM THAT POINT.

Right here it may not be out of place to insert another difficulty, with which we have to deal when looking for the original mother focus of cheesy degeneration after death by acute tuberculosis. The original focus may be nothing more than a corpuscular one (Waldenburg); and whether existing within as a remnant of inflammation, or having been introduced from without, it may have lain dormant, and with its local inflammatory products remained unabsorbed, owing to the absence in its immediate neighborhood of a good and unobstructed absorbent. Circumscribed encapsulation may have taken place. Thus it may remain innocuous until some general disturbance of the health takes place rendering the patient less capable of resistance. I have mentioned before my belief that, as soon as fever sets in, the oft-mentioned "breaking-down" of the quality of the blood in such a patient rests upon *the cessation of the formation of its constituents* (*notably red blood-corpuscles, and perhaps fibrine*), *as evidenced by enlargement of the spleen, etc.*, and not so much in the loss of any constituents already existing. This, as you know, would relieve the absorbent lymphatics and veins from assisting in the process of assimilation; their whole attention may be turned to the absorption of home tissues and products. It is at such a time of vascular activity and rapid absorption that such an isolated cheesy focus may readily fall a prey and be carried into the general circulation. First, its molecules are carried to the right side of the heart in constantly enlarging vessels, and, passing into the pulmonary circulation, reach smaller peripheral branches, where they may become lodged or be deposited in the lung; which fact would readily enough account for the predilection of tubercle and the development of consumption in the lung and equally as much for the pleura, when we recall the fact that the "branches of the pulmonary artery pass directly into the pleura" (Hyrtl).

This, too, would give color to the before-mentioned "embolic theory" of Waldenburg and others, as yet, however, but conjecture.

Again, where such active absorption is going on, is it to be

wondered at if the whole of the cheesy focus be absorbed, so that on autopsy we find naught but tubercle of the same crop and degree of development?

In addition, I merely hint at the difficulty of searching every nook and corner of every tissue of a cadaver for remnants of a cheesy parent focus.

I have dwelt at such length upon the relations of pleurisy to tuberculosis, as its importance cannot be overestimated.

Lastly, there remains for our consideration the relation of *peribronchitic processes* to tuberculosis. Here, so far as the cirrhotic conditions are concerned, the same holds good in the connective tissue of the bronchi, as has been laid down for that of the lung parenchyma. Part of these processes are the basis for Rindfleisch's elaborate explanation of the progressive stages of tuberculization. In order to avoid confusion and put things as clearly as possible, I will devote part of the chapter on phthisis proper to Rindfleisch's deductions.

RELATION OF TUBERCULOSIS TO SCROFULOSIS.

For the past century there has existed a clinical premonition, to the effect that "tuberculosis is developed from scrofulosis." A mathematical formula with two unknown factors, a deduction without major or minor premise, it has stood until, in the last twenty years, these factors have been investigated and elucidated.

Pathological anatomists (Virchow, Buhl, Reinhardt) as well as clinical observers (Niemeyer, Traube, and others) brought it into such shape that distinct questions could be formulated, to be answered by experiments on animals. Villemin (Études sur la Tuberculose) paved the way to systematic experimental investigation, and was followed by Lebert, Colnheim, Langhans, Fraenkel, Waldenburg, and others. They have not succeeded in closely defining either tuberculosis or scrofulosis, but have considerably narrowed the sphere within which both are permitted to exist.

You have seen what tubercle is, under what conditions it is formed, and in connection with what other conditions it is likely to supervene. If, now, I will endeavor to prescribe and limit what constitutes scrofulosis, you will be prepared to understand the relation between the two.

Against noxious agents, which excite inflammatory action, which surround us and which we meet during life, different individuals possess very different conceptions. The greater number of these noxious materials may be regarded as being suspended in the air; and against their absorption into the human body the thick epithelial layers of the skin are the physiological safeguard. If you now imagine such an impervious covering somewhat imperfectly developed, and even defective in its unbroken continuity, you are quite near the truth. Between the epithelial and epidermic cells minute porous canals are found, and it is especially in the Rete Malpighi that they are easiest demonstrable. Nothing is more likely than that these canaliculi are of varying width and more or less developed in different persons, and that persons with open, easily accessible pores are most prone to oft-repeated entrance of noxious matter. Such persons are called "irritable" and "easily vulnerable," and are justly supposed to be likely

victims of scrofulosis. And we can safely place on record, as our first proposition, this dictum: THE PREDISPOSITION TO SCROFULOSIS IS SHOWN BY THE INCREASED VULNERABILITY AND GENERAL IRRITABILITY OF THE BODY, AND A TENDENCY TO REPETITION OF ORDINARY INFLAMMATORY PROCESSES.

But, since not every irritable person is scrofulous, but all scrofulous persons vulnerable and irritable, we must add another quality to such inflammations. It is, that *inflammations of this kind have a great tendency to extend themselves, both as to space and time.*

They are never sharply defined nor confined to one locality, but show a tendency to spread sluggishly over surfaces and into organs, and by their sluggishness to become chronic, to extend past the ordinary time-table of inflammatory processes.

We next find that these processes depend for their extension upon the absorbents of the plasmatic circulation, upon the lymphatic vessels. The porous canaliculi are part of the lymph-carrying system, and take part in carrying movable connective-tissue cells, migrated white blood-corpuscles, as well as any noxious matter from without. If we may imagine the lymphatics proper to be as voluminous in proportion as are the admitting pores, the explanation for ready infection would be found. No positive examination has as yet been made regarding the comparative size of lymph-vessels in different persons. Instead, we recognize a great difference between the dry connective tissue, poor in cellular elements, of the adult, and the moist, juicy connective tissue, very rich in cells, of the infant. Scrofulous children present a "pasty," pulpy appearance. This is due to the over-filling of the connective tissue with nourishing juices, plasmatic fluid. The infant, whose growth is as yet to follow, is naturally supplied with an abundant plasmatic circulation, with larger calibre of vessel. Its growth is dependent upon rapid assimilation and equally rapid retrograde absorption. Hence the readiness with which noxious material is absorbed in childhood. Let the noxious matter enter the dilated canaliculi in force, and the lymphatic stream will quickly carry it along to the neighboring glands.

Thus we have our second depot, and may formulate this fact as follows: INFLAMMATION OF LYMPHATIC GLANDS IS THE SECOND IMMEDIATE STAGE OF SCROFULOUS PROCESSES. At best all

inflammations of lymphatics run a more or less chronic course, as you will remember when thinking of syphilitic and other infections. Recall to mind what I have twice stated in previous letters, that slow inflammatory processes do not tend to acute œdema, suppuration, and destruction, but rather to the formation of connective tissue. An exception to this rule has been claimed by Virchow to exist in the formation of minute quantities of pus in a lymphatic vessel previous to its entrance into a gland (vas efferens), where the fibrillary connective tissue is almost wanting and cells abound. Billroth, and after him Hueter, holds to the belief that cells and lymph or plasma are the only constituents to enter the gland, and that at no time did the cells form part of an abscess.

The next step in the fate of the infiltration of the gland is the caseous metamorphosis of the cells. This, for the present, ends the cycle of events. The beginning is a local inflammation (pharyngitis, rhinitis, eczema) correctly enough called scrofulous inflammation, if you will but keep well in mind that this inflammation in its etiology is an ordinary inflammation excited by the same ordinary noxæ as in common inflammation. But the difference is that they run their course in *tissues so constructed as to permit of the inflammation becoming chronic, and of prompt conveyance of the noxious matter into lymphatic glands.*

Our third station, then, and for a time a halting-point in the course of scrofula, is this: SCROFULA CULMINATES IN CHEESY INFILTRATION OF LYMPHATIC GLANDS.

With this present ending of local manifestations begins the danger for the whole organism. To understand this, let me relate the possible ultimate fate of these cheesy degenerations. Resolution is a bare possibility, but not probability. The mass is cut off from further connection with absorbent lymphatics, and thus might be looked upon as dead, were it not for the indirect nourishment it receives from the circulation. Complete drying up of the mortar-like mass of cells and encapsulation are possible, but not likely sufficient to be a permanent protecting investment. And why? Because the same locality (skin or mucous membrane) which permitted of the passage of the infection formerly, will be likely to do so again, and get into sufficiently close contact with the cheesy mass to light up a *subacute suppuration in the caseous mass.* Such pus is mostly found in the most super-

ficial layers of the gland—an additional proof that the irritation culminating in suppuration came from without.

Self-infection in the course of general or local diseases may bring toxic material within reach of the cheesy mass. But this need not be dwelt upon, inasmuch as all this will be comprehended in the explanation I am now about to make, of how tuberculosis springs from scrofulosis.

Not only does scrofulosis beget in many this general disposition to inflammation running the course described, but, like other diseases, *furnishes the inoculating virus for miliary tubercle.* In the experiments on animals, where the infecting detritus comes from without, the blood circulation must be looked upon as means of transportation, and the deposits observed in connection therewith must have their origin in the small arteries or capillaries.

In the formation of tubercle in man, it is most frequently the lymph tract which takes up the material and carries it on to general distribution.

In accord with this, we surely enough do find *tubercular formation in the immediate vicinity of cheesy deposits, along the track of lymphatics and in lymphatic glands*, into which they pass.

In what manner a general infection comes about, and acute miliary tubercle is developed, is not so easy to say. Probably something does finally pass into the blood; but what takes place here, whether merely physical or also chemical changes occur, or both, is at present not settled.

I have before touched on Rindfleisch's opinion that tuberculosis, and for that matter consumption in any form, is on a scrofulous basis, and will give an outline of his views when finally reviewing phthisis pulmonalis.

What I will quote here is part of what he says in regard to the existing relation: "The final disintegration of a scrofulous infiltration is effected by chemical metamorphosis, which, aside from very small fat drops and albuminous granules, which can be seen by the microscope, furnishes an indefinite quantity of soluble substances not visible microscopically. All these substances, however, not only may, but must be reabsorbed. When we see that they are just scrofulous persons who are the favorite victims of tuberculosis; that we know no such thing as tubercu-

losis of non-scrofulous individuals; when we see that all the various pictures of disease constituting tubercular phthisis are composed of scrofulo-inflammatory elements on the one hand, and tubercle formation on the other; when we can correctly say that scrofulous individuals need only to have an inflammation to be at once in danger of tuberculosis,—every one will look for the reason of these facts in the peculiar course and termination of the primary inflammation, which, in its sluggish cheesy infiltrations and purulent secretions of mucous membranes, generates the poison itself, which, taken into the juicy parts of the body, produce tuberculosis. This sentence contains all we need to know for the comprehension of the intimate relations between scrofulosis and tuberculosis."

We may then elucidate one of the final terminations of scrofulous products, as well as clinch the relation of scrofula to tuberculosis, by the following formula:

THE LINGERING CHEESY INFILTRATIONS IN LYMPH-GLANDS AND THE PURULENT SECRETIONS OF MUCOUS MEMBRANES GENERATE A VIRUS, THE EXACT NATURE OF WHICH WE DO NOT KNOW, WHICH, CARRIED ALONG BY THE EFFERENT LYMPHATICS, DEPOSITS AS TUBERCLE.

How does the virus "deposit" as tubercle?

This seems to be the proper place for referring once more to the embolic theory of distribution. Thus far it has not been proven, but from all observations it is likely that the granular detritus is carried through the right side of the heart into the lungs and into the most minute vessels; that at length they become clogged in some remote vessel as emboli; that white blood-corpuscles migrate from the blood to the surrounding tissue, and thus miliary tubercle is formed.

This would seem to call for emboli of respectable dimensions to fit the lumen of capillary vessels. The lesser granules may pass on, partly to the pleura, and stagnate only where capillary circulation happens to be a trifle sluggish, or, for aught we know, they may migrate and become imbedded in connective tissue.

Miliary tuberculosis would then be counted as an embolic disease, a pyæmia with multiple inflammatory centres.

Indeed, the arranging of tubercle along the walls of capillaries, most frequently at a bend in their course (Billroth), and lastly the presence of one or two stellated giant-cells in the centre of tubercle (Langhans), all point to the embolic origin of tubercle.

Of course, in order to be carried along in the circulation, cheesy detritus must first be absorbed by the lymphatics; and this too is in accord with clinical observation, which teaches that suppurative disintegration of the cheesy lymphatic gland as a rule precedes the development of tubercle. We have thus a reabsorption of a cheesy mass; and while we may not conclude to believe, with Rindfleisch, that its disintegrated constituents "must be absorbed," the possibility and probability of a total absorption is given.

As a final proposition, let me say: NOT DETRITUS OF CASEOUS LYMPH-GLANDS CARRIED INTO THE CIRCULATION ALONE MARKS THE RELATION BETWEEN SCROFULA AND TUBERCULOSIS. LOCAL SCROFULOUS INFLAMMATIONS GIVE RISE TO THE FORMATION OF TUBERCLE IN THE GRANULATING SURFACE ON THE SPOT. The most lucid example of this local development is found in synovitis fungosa, where Koester demonstrated tubercle in the granulation tissue of a suppurating inflammation of the joint.

This is in accord with what I have endeavored to show you when speaking of the formation of tubercle after pleurisy. To this I refer you, and will add that another example of local tubercular infection, in which we are interested, is its occurrence in caseous pneumonia, where miliary tubercle follows into the periphery of the pneumonic cheesy mass. All these facts have been dwelt upon when speaking of local and general tuberculosis.

PHTHISIS PULMONALIS.

The term "consumption" comprises all *progressive destructive processes of the respiratory organs*, which do not occur immediately in the train of acute inflammation. It is accompanied by more or less rapid *wasting and reduction in weight of the body*, and one of these two factors is likely to predominate. We may say that the more rapidly phthisis runs its course, the more rapid is the destruction of tissue; while the slower the progress, the stronger will it be marked by wasting and reduction of the body.

PHTHISIS IS THE COLLECTIVE EXPRESSION, THE HIGHEST POTENCY OF A NUMBER OF ACUTE INFLAMMATORY PROCESSES WHICH HAVE FAILED TO RUN THEIR PRESCRIBED COURSE.

The termination of such processes is when, having passed into the chronic condition, their products become caseous (cheesy pneumonia), while the peripheral connective tissue endeavors by proliferation to form a capsule for the cheesy mass, and, finally, by contraction and almost total exclusion of all nutritive material, to encapsulate and render it harmless.

Where this is not accomplished, the cheesy mass may undergo a subacute suppurative process; tubercle may be formed on the spot; and the active neighboring lymphatics absorb and carry to other parts the granular matter and tubercle.

What agency is first concerned in the formation of tubercle or the mortar-like, cheesy mass, we do not know to-day, any more than formerly.

Consumption, then, may be said to *begin where cheesy degenerations in the lung are lighted up to renewed activity.*

Referring again to the factors of destruction and wasting, we may formulate the progressive processes into two principal heads, with a few subdivisions:

1st. Where a rapid course tells of the equally rapid destruction of the respiratory organs, where, in short, the disease may be said to be acute.

a. Acute miliary tuberculosis, disseminated throughout the lung.

b. Inflammatory conditions, which do not lead to sudden and immediate death, as do gangrene and purulent infiltration. Under this head belong acute pneumonias and purulent peribronchitis.

2d. Where a slow course tells of equally slow destruction of the tissue and attempts of nature at repair.

a. Caseous pneumonia (cheesy remnants of inflammatory processes, both lobar from pneumonia and lobular).

b. Chronic interstitial pneumonia (cirrhosis, with the formation of bronchiectatic cavities) as a one-sided, independent disease.

c. Local proliferation of connective tissue (peribronchitis nodosa), or in the periphery of cheesy masses, endeavoring to form a capsule around it.

3d. The combination of two or more of the above conditions. We find acute tuberculosis supervening upon the results of acute inflammatory conditions, with cheesy pneumonia, with chronic interstitial pneumonia, and, *vice versa*, see an infiltration of tubercle becoming cheesy, always beginning in the centre of its nodules, and the development of connective tissue in the vicinity and its subsequent contraction. The tubercular and inflammatory elements are mixed, and pursue their course together, unfortunately rarely towards repair, but towards destruction.

I have before intimated that several observers have recently fallen back upon the old tubercular theory, not with all its defects, but merely endeavoring to demonstrate that all cases of consumption are *tubercular, on a scrofulous basis*, and that inflammatory conditions only occurred as accompanying elements. Rindfleisch is the chief exponent of this theory; and in giving an outline of his views, I will redeem my promise of the foregoing chapters:

In scrofulous products, we note especially their *great tendency to cell-development*, and an equally great one to *disintegration of this same hyperplastic cellular element.*

White blood-corpuscles, which in normal individuals pass from vessels of an inflamed tract to an adjoining surface and into lymphatics or lymph-glands, or else collect in abscesses, will in scrofulous persons become *larger* on their way through the connective tissue, by becoming impregnated with albuminous substances, which substances are the direct cause of their slow degeneration.

In scrofulous catarrhs we note an abundance of cells, and the thick, quickly-drying character of the catarrhal secretions. Cells likewise abound in the submucous tissue, from where they wander to the surface, become granular, and at the same time fattily degenerate, or else they pass into lymphatic vessels. Their detritus becomes mixed with lymph, which flows off from the inflamed tract into the lymphatics. Whether or not we here have the formation and transportation of tubercle virus, we do not know.

Tuberculization of scrofulous persons. Final disintegration of a scrofulous infiltration is accomplished by chemical metamorphosis, furnishing minute fat-globules and albuminous granules, together with an indefinite quantity of soluble substances, the latter not visible microscopically. All these substances not only may be, but *must* be reabsorbed. Thus scrofulosis, with its lingering cheesy infiltrations and purulent secretions of the mucous membranes, *generates its own poison*, which, taken up into the juices of the body, develops tubercle.

Those who look upon tuberculosis as a purely independent acute infectious disease, should remember that the tubercle virus in question is self-manufactured and the infection a self-infection. Nor has this virus ever been proved capable of inoculation upon perfectly healthy persons. Rindfleisch's views may therefore be thus summarized :

Tuberculosis hardly ever occurs except in scrofulous persons.

Tubercular phthisis is nothing but a combination of scrofulous inflammation and tubercle.

In scrofulous persons any inflammation brings with it the risk of tuberculosis.

The well-known cheesy intumescence of lymphatic glands has always been looked upon as the chief product of scrofula. And indeed the lymphatic glands are the *anatomical centre of scrofulous inflammation and hyperplasia.* Scrofulous glands are always to be looked upon as tuberculous glands. The swelling and obstruction of the efferent lymphatic may cause the disease to remain localized for a time in the gland.

The course pursued by scrofulo-tuberculous troubles justifies a division into primary, secondary, and tertiary tuberculosis, if you will only not apply these appellations too strictly, but only as a generalization. In accordance with the various "depots," of

which I spoke in discussing the relation of scrofulosis to tuberculosis, we may divide the development of tubercle into three stages also:

By primary tuberculosis we comprehend *local affections of the various organs* of the body, which happen to be affected by scrofulous inflammation and development of tubercular elements on the spot.

Secondary tuberculosis designates the *tubercular infiltration of lymphatic glands.*

In tertiary tuberculosis, finally, we have the *dissemination of tubercle throughout the organism*, affecting organs not primarily diseased, as, for instance, the lungs, liver, kidneys, and the serous membranes.

The stages, in short, are: *strictly local*, whether on surface (skin and mucous membrane) or in internal organs; *lymphatic glands; distribution* throughout the whole organism.

BEGINNING OF LUNG TROUBLE.

I.—*Chronic Tuberculosis.*

An old maxim teaches that a lingering catarrh gives rise to consumption. The truth of this is: when a case of catarrh of the apex is diagnosticated, an autopsy would, in addition to catarrh, reveal genuine tubercle upon the acini of the lung parenchyma supplied by the affected bronchi. Rindfleisch believes that the catarrh is the earlier, tuberculosis the later manifestation. He imagines the tubercle virus to be contained in the cellular catarrhal secretion of anæmic, scrofulous individuals. You know that other authorities hold that the primary presence of tubercle incites to catarrhal inflammation. As usual in such questions, the truth is most likely in the middle, that is, both cases are possible, and the history of the case should solve the question in each individual case.

The points at which the smallest bronchioles become continuous with the acini of the lungs are the situation of the first eruption of veritable tubercle.

1st. TUBERCULOUS INFILTRATION OF ALL EDGES AND PROJECTIONS, which exist at this point of junction, is the first pathological change, and leads to the formation of circumscribed white

nodules in the connective tissue. This corresponds to Laënnec's tubercle-granulation, and is the anatomical nucleus of the disease. With its softening particles in the centre (if any exist), a broad zone of cheesy infiltration and peripherally a zone of recent large cells, it represents an infiltration of the connective tissue of the lung, bloodless, incapable of suppuration, reabsorption, or organization, but only of degeneration.

Initial hæmoptysis and pleurisy. Before considering the further progressive mission of the lung trouble, the above conditions must again be touched upon, because it is at this stage that their occurrence furnishes a clew for diagnosis.

The minutest branches of the pulmonary artery take part in the tubercular invasion. A pushing on of perivascular hyperplasia into the inner coats of the artery takes place, and we have a tuberculous degeneration of the middle coat as well as of the intima. A sudden tension by muscular action, or temporary overfilling of the artery, may lead to rupture and hemorrhage. I have, in a previous chapter, said that hæmoptysis is likely never the beginning of lung trouble, but the result of conditions prepared some time beforehand. No satisfactory dissections have been made immediately following a hæmoptysis.

Pleurisy, you are aware, is an accompaniment of all stages of consumption. The adhesive form is the rule. The relations between tuberculosis and pleurisy have been discussed in the chapter on "Tuberculosis." Rindfleisch states that while tubercular pleuritis is not rare, the pleuræ of a consumptive patient rarely shows tubercle. On the other hand, he draws attention to the great wealth of blood and of vessels in all its products. I have told you of the passage of small branches from the pulmonary artery into the pleura direct. This would make the pleura a sort of reservoir in case of stagnation in the lung, while the presence of a large quantity of blood of sluggish movement would account for the uncommon development of new connective tissue which takes place in the pleura.

2d. *Second Depot of Consumption.*—Caseous bronchopneumonia. From the tubercle-granule there arises, after a time, the larger cheesy mass, formerly known as crude or yellow tubercle. The cheesy mass passes from the above-mentioned bronchial endings into the smallest bronchi and further on into all the branches, the shape of the cheesy mass corresponding to

the course of the involved bronchi up to bronchi with cartilage rings, where its progress ceases.

From this on, the further progress may be looked upon as consisting of three factors, each more or less independent:

1st. Tuberculous ulceration of bronchial mucous membrane.

2d. A nodular tuberculous peribronchitis.

3d. Scrofulous cheesy pneumonia, so-called desquamative pneumonia.

Observe that Rindfleisch thus includes all cheesy and cirrhotic processes, which by others have partly been accredited to remnants of acute inflammation and by Buhl to the products of desquamative pneumonia and connective tissue hyperplasia, and gives the parentage to tubercle alone.

Third Depot.—a. Tuberculous ulcers of mucous membrane have a tendency to spread towards the periphery as well as into the depths of tissue. In the periphery we have the same nodules of tubercular infiltration which compose the primary tubercle. In the depths we find the eruption of large miliary tubercle at the bottom of the ulcer and at a little distance in the deeper strata of the bronchial walls. They are in close relation with the lymphatics, being in the walls of such a vessel, or they may surround the lumen of such lymph-vessels as lead downward into the submucous and peribronchial connective tissue.

b. Peribronchial infiltration consists of the propagation of tubercle in the peribronchial lymphatics. Almost one-third of an infiltration consists of aggregated tubercle, the other two-thirds of connective tissue in the process of contraction. From this second depot of peribronchial invasion there is, again, direct continuity between it and the third factor, cheesy broncho-pneumonia, or exquisitely scrofulous desquamative pneumonia.

c. Desquamative pneumonia. The desquamation and change in form of alveolar epithelium, which Buhl thinks of sufficient importance to present as an independent picture of disease, and which by others is included in the catarrhal form, is in reality a stereotyped accident of most disturbances which befall the parenchyma of the lung, and merely act as an accompaniment to the more important changes in the connective tissue. This is infiltrated with an enormous quantity of cells, so that connective tissue, elastic fibre, and vessels can no longer be seen. Compression of the capillaries next begets bloodlessness, leading of course

to lack or nearly total absence of nutrition from the anæmic parts, and to cheesy degeneration.

Before caseous metamorphosis becomes complete, contraction of the accompanying connective tissue takes place. The degree to which desquamation of alveolar epithelium supervenes, and the independence of its occurrence in connection with peribronchial infiltrations, varies somewhat, and by its course justifies us in distinguishing between a chronic and a more acute progress of consumption.

Fourth Depot.—Common chronic form of consumption may be said to occur where the scrofulous infiltration of peribronchial and perivascular connective tissue keeps on continuously to invade the adjacent connective tissue of the parenchyma, the connective tissue most intimately connected with the lung tissue proper, and bearing the same relation to the peribronchial tissue from whence comes the invasion as does the connective tissue of the muscles bear to the subcutaneous layers. The desquamative pneumonia here appears the least independent, serving only to assist in augmenting the fibro-cheesy mass, and by uniting with other parts similarly infiltrated.

The difference in the progress of peribronchitic infiltration and the desquamative process is that the former passes upward from bronchiole to bronchus, while the latter's invasion is lateral into the parenchyma. What now occurs, when the lumen of a bronchiole is thus occluded, is that atelectasis supervenes. A corresponding portion of the lung will contract by its own elasticity to its smallest possible compass, the air within it disappears, and is replaced by a certain amount of serum and largely by capillary nets from its own walls. One would think that here local death would supervene; but, on the contrary, the next step is a passive hyperæmia, by which the atelectatic part becomes œdematous. Serum is effused from the dilated vessels filling the alveoli, and distending them. By gradual thickening of the serum, the infiltration may assume a gelatinous appearance. To this is added fatty degeneration of the alveolar epithelium.

Thus, in cases where the lesser and most minute branches of the bronchial tree have fallen a prey to tuberculosis, we have in the affected lung circumscribed sections, with atelectasis, inveterate and more recent œdema, and fatty degeneration of the epithelium.

A different picture will present itself where a somewhat reduced but still open circulation encourages rather than retards the development of the desquamative process, and the latter assumes more independence. This may occur to such a degree as to involve an entire lobe in a diffused inflammation; but this lobe must be one previously invaded by tuberculosis.

Oftenest it occurs as a fresh onslaught, where cavities already exist at the apex.

True to his scrofulous theory, Rindfleisch goes on to say that—

1st. The relation of desquamative pneumonia to tuberculosis proves it to be of scrofulous parentage likewise; and,

2d. Its independent occurrence where tubercle already exists stamps it in a measure as an independent disease, such as Buhl claims it to be.

But, according to these two factors, *desquamative pneumonia is a specific scrofulous inflammation*, and as such to be classed with all scrofulous catarrhs—that is, common inflammation in an individual prone to the development of cheesy material.

Fifth Depot.—Formation of cavities. The term "cavity" should be restricted to all holes of larger size which originated in necrosis and ulceration of lung parenchyma; "bronchiectasis," on the other hand, denoting the distention of previously existing hollow tubes. In bronchiectatic conditions the pressure of air, failing to impart itself to the occluded parenchyma beyond, exerts its power on the bronchial tubes. The resisting power of the lesser bronchi is none of the greatest, and many a bronchiectasia can compete as to size with large cavities.

The earliest stage of formation of true cavities may be said to exist in the little spots of softening, which we occasionally find in the interior of tubercle granula, and which spots correspond to the former lumina of the alveolar passages. In the further progress of disease and the formation of cheesy nodes the lesser bronchi likewise represent the plan of the future cavity.

The softening masses afterwards empty into the cavity, *through the mechanism of respiration.* The wall of the bronchus, inflamed, infiltrated, and hyperplastic, is rendered more yielding, less able to withstand extensile traction by the muscles of respiration than the surrounding parenchyma. Hence the bronchus dilates and the parenchyma contracts.

In the process of emptying of the contents of a cavity, the forces of inspiration reach the periphery of the softened mass. Thus cavities are formed by the air penetrating into softened material and by creating a hollow space in forcing its discharge, and not by the emptying of cavity contents into a bronchus.

Cavities are to be regarded as spaces filled with air and acted upon by the forces of inspiration. The enlargement of a cavity is finally checked by reaching the pleura, which becomes thickened and *forms a definite limit.*

Other modes of enlargement are the junction of two cavities and the further drawing in of dilated bronchi into the walls of the cavity.

Mixed and rare varieties of consumption. We have seen ere this that the post-mortem appearances in consumption are exceedingly varied, all processes sometimes being represented in different stages of development and different as to relative quantity. We may find tubercle-granulation and peribronchitis, gelatinous infiltration (as remnant of catarrhal products) and cheesy pneumonia, catarrh of the bronchi, atelectasis, bronchiectasis, miliary tubercle and tuberculous ulceration, pleuritis and hyperplasia of connective tissue.

Yet some one process will be found to predominate, and the order of events may be found out with the aid of the history of the case.

Still Rindfleisch and others draw attention to a few forms which deserve special mention.

Of such is the uncommon form in which phthisis presents itself in children under five years of age. As in their physiological condition, so in pathological processes, there is an increased tendency to shedding of epithelium; in the course of inflammations we find a predominance of desquamated epithelium in the products. This is true of catarrhal pneumonia (capillary bronchitis), and all the more so in phthisis, which is a compound of scrofula and tuberculosis. Each individual tubercle, indeed, is found to be surrounded by a zone of desquamative pneumonia.

In the adult, one of the modifications in phthisis consists in the prominent *participation of the lymphatic system.* The fact has been dwelt upon before that the lymph-vessels are a favorite site for tubercle; and I refer you to the chapter on Acute Interstitial Pneumonia for the facts bearing upon the inflammatory

part of it. Rindfleisch believes that this form represents syphilitic tuberculosis. It is found in the lower lobes, together with hyperplasia of connective tissue and cirrhosis (*vide* chapter on Acute Interstitial Pneumonia), and the tubercular eruption of the lymphatics is likewise accompanied by the slow desquamative process.

II.—*Acute Tuberculosis.*

Referring back to the beginning of Rindfleisch's propositions, we distinguished three stages: 1st. The local scrofulo-tubercular primary affection. 2d. Tuberculosis of the lymphatic glands. 3d. General miliary tuberculosis.

General miliary tubercle is pre-eminently a constitutional disease, which, in the number, size, and distribution of the erupting tubercle, finds only an imperfect expression of itself. This is owing to the rapidity of its course, giving the successive tubercular eruptions barely time to degenerate before death ensues.

Rindfleisch consistently looks upon this disseminated *miliary tubercle as a circumscribed centre of scrofulous inflammatory infiltration*, and says that neither giant-cells, nor the fact of tubercle being bound to the minutest ramifications of the pulmonary artery, can change its character as a minute inflammatory product; while in scrofulous persons it represents a small centre of scrofulous inflammation.

Thus far Rindfleisch. I have given his views at some length, as representing as nearly as possible the views of several authors who hold that consumption can only spring from inflammatory products of scrofulous persons; that tubercle is one of these products; and, finally, that consumption exists only as "tubercular phthisis."

My next endeavor will be to condense the views of such authors as look upon consumption as a PRIMARILY CHRONIC INFLAMMATORY PROCESS, AS WELL AS THE FINAL DESTRUCTIVE PROGRESS OF CHEESY REMNANTS FROM INFLAMMATIONS OF THE LUNG, which have gone before and run their course, with more or less accompanying cirrhotic processes, and the occurrence of tubercle, either secondarily or more rarely as an entirely independent disease.

This review will represent the opinions of leading pathologists, several of whom (Ruehle, Juergensen, Fraenkel, Waldenburg) have been quoted before.

Phthisis is anatomically a chronic inflammatory disease, with intercurrent simple forms of inflammation, which latter may heal by cicatrization. Such limitation and local healing is effected by simple inflammation in the surrounding parts.

For the sake of a thorough understanding of our subject, I find it necessary to dwell briefly on the etiology of phthisis. Time and space will not permit of mentioning every possible cause; a few salient points only will be noted.

Everything which tends to diminish quality and quantity of the blood, which depresses the nervous system, all agencies by which the balance between waste and supply is destroyed, will render an individual what is commonly called "sickly." Now when the circulation is disturbed, whereby nutrition is lowered and peripheral circulation becomes insufficient, or when, through central or peripheral agencies, motion or sensation is impaired for more than a limited period, we may suppose the afflicted individual to be very vulnerable. General vulnerability (as you will remember from the chapter on Scrofulosis) is closely allied to scrofulosis, not by covering all the characteristics of the same, but because it may be the starting-point for scrofulous inflammation, which in an adult may not have occurred since infancy.

Imagine, for example, a disturbance of circulation or innervation, caused by a gastric catarrh, to remain longer than they are found as a rule, and we have general vulnerability, which in turn gives rise to oft-repeated and dragging bronchial catarrhs or other inflammations characterized by active cell-proliferation. From scrofulosis to the development of tubercle is but a step.

I have touched upon this cycle of events in order to once more impress upon you the importance of bronchial catarrhs as well as inflammations of the lungs. We have, at one end of our string, inflammation (generally catarrhal) and localized cheesy centres, which may have lain dormant for years, especially in lymphatic glands, and, at the other end, consumption and tuberculosis. Now whether in any given case the *catarrh*, as most immediate product of general vulnerability, a previously existing *cheesy centre*, or a recently developed *local tuberculosis*, was the first to be in the field, is so difficult of demonstration as readily

to account for all differences of opinion on that point as well as the absence of proper statistics.

In this connection we should not omit to remember one cause, if not the chief one, of disturbances of circulation in the lung. For twenty years Dr. Brehmer, of the Sanitarium for Consumptives at Goerbersdorf, has held that the phthisical habitus was characterized by *smallness and flaccidity of* the heart. This theory was first put forth by Rokitansky in connection with other appearances. Brehmer, however, insisted upon its importance, and looked upon it as the chief etiological force of Consumption. Of late, however, he has become convinced that he had overestimated the importance as well as the frequent occurrence of small hearts.

Any disturbance of the equilibrium between waste and supply, as you have seen, leads to conditions favorable to that final collective expression of all chronic nutritive changes, to consumption. If the waste be too great from any cause, the blood will suffer in quality as well as quantity, and among the organs to suffer by this, and to become bloodless and flabby, is the heart. The heart, whose contractive duties require constant and sufficient nutriment to keep up its propulsive powers. Given a person with a naturally small heart, it may not require any great length of time to weaken its power of propulsion. In the lungs the result of excessive waste from any cause would be seen in the occurrence of anæmia of its tissue and consequent loss of its elasticity, thus giving rise to that most important factor in consumption, a paralytic lung.

If the supply be excessive, the primary result will be a clogging up in the lymphatic system, and as a natural consequence followed by stagnation in the circulation of the blood; and nowhere more so than in the lung, whose capacity for blood is so much greater than that of other organs. Here, again, the effects will be found to consist of loss of elasticity, of dilatation (emphysema, bronchiectasis), and what is of more importance, to deposits (infiltrations, tubercle). In the heart again, stagnation acting backwards upon it will not necessarily produce hypertrophy and more propulsive power, though the first effect may be such; but eventually flabbiness must result.

Taking these causes together, we may briefly sum up thus: ABNORMAL SMALLNESS AND FLABBINESS OF THE HEART, WHETHER

CONGENITAL OR AS A RESULT OF PERIPHERAL DISTURBANCES REACTING UPON IT, IS A CONDITION FROM THE EXISTENCE OF WHICH WE DATE SUCH LESSENED AND RETARDED NUTRITIVE CHANGES IN THE LUNGS, CAUSED BY INSUFFICIENT SUPPLY OF BLOOD, AND ITS CONSEQUENT STAGNATION FROM LACK OF CENTRAL PROPULSION AND PERIPHERAL ELASTICITY, AS WILL MANIFEST THEMSELVES BY PRODUCTS WHICH WE CALL RESPECTIVELY: HYPERPLASIA OF CONNECTIVE TISSUE, CHEESY DEGENERATIONS, AND TUBERCLE.

I regret that my plan will not permit me to go again over the ground of the relations of various inflammations to consumption. As it is, I refer you to each individual chapter, and only repeat that, as a general thing, this dogma is still entertained:

INFLAMMATORY PROCESSES OF THE LUNG WHICH FAIL TO COME TO A RAPID RESOLUTION AND WHICH LINGER FOR MORE THAN A VERY LIMITED PERIOD, ELABORATE PRODUCTS, WHICH THROUGH CASEATION LEAD TO PHTHISIS.

You are aware that nearly all cases of incipient phthisis begin at the apex, and oftenest indeed without the previous occurrence of an acute lung trouble. You hear and read of "catarrh of the apex," and want to know what pathological conditions transpire under that name. In the first pages of this chapter I attempted to lay out a schedule. An additional one might be made out on the general basis of etiology:

1st. Those affections which are the sequel to previous acute lung trouble.

2d. Where the indirect cause is general vulnerability following previous disease other than lung trouble, frequently manifested for a time by nothing but a *gastric catarrh as indication of a disturbance of nutritive changes.* As soon as râles can be heard at the apex, we are confronted by the question which of the three following conditions, or their composition, had first existence:

Local proliferation of connective tissue and induration. (See chapter on Chronic Interstitial Pneumonia.)

Caseous deposit, either in a scrofulous gland from infancy or other cheesy remnants, which collectively we call caseous pneumonia.

Local tuberculosis, either in a cheesy centre or independently, having originated from some purulent surface; as yet, however, strictly localized.

Two or all three of these conditions combined. Such local inhabitants may, at first, produce no appreciable symptoms. Catarrh of the lesser bronchi supervenes, and now the process can be diagnosticated. Observe that I make the catarrh a secondary process, and remember that, on the other hand, there is no positive proof that a bronchial catarrh may not spread to the parenchyma and create catarrhal pneumonia. By far the majority of catarrhs, however, does nothing of the kind.

The further progress of the "catarrh" is marked by copious desquamation of epithelium, which, together with the tough and equally copious secretion, forms the products, which undergo cheesy degeneration and destruction. I lay stress upon "desquamation," as this forms a connecting link with Buhl's theory of desquamative pneumonia, which he insists begins at the apex, in contradistinction to catarrhal pneumonia, which begins at the base. Remember, also, that secretion and desquamation are equally as well dwelt upon by other authors in connection with apex and other catarrhs; but they do not acknowledge the necessity of having a separate designation for such processes.

What has just been said of "catarrh of the apex" is intimately associated with what now remains for me to say of the pathology of phthisis.

If we were permitted to use words in the ordinary meaning as technical terms, we could safely say that consumption was but a termination of various diseases of the respiratory organs.

Once any process has passed its initial stages, it rarely remains unmixed with other processes, one often passing into and becoming lost in the other. Thus it has been my endeavor to present to you the chief types of chronic lung trouble, and to constantly remind you that they very frequently combine in the same individual or follow upon one another.

That the whole field may appear quite clear to you, I will not condense the various processes in any further schedule, but once more explain the whole range of consumptive troubles in the plainest terms possible. Repetition is, of course, unavoidable.

Beginning with *acute miliary tuberculosis*, we find in it a special affection, an independent infectious disease, which runs a rapid course, does not undergo various stages of progressive de-

struction, nor does it kill by consumption. It differs from other acute infectious diseases by being self-infectious, the virus originating from some previously existing cheesy centre. Tubercle produced by inoculation from without are not identical in man with miliary tubercle, but are minute centres of lobular pneumonia. What the chemical or other changes are by which cheesy matter, etc., becomes and is deposited as tubercle, we do not know. The white blood-corpuscles, new connective tissue cells, endothelial cells, these three factors of growth and repair have been credited with the parentage. Likewise any "corpuscular matter" (Waldenburg) causing minute emboli in the capillaries of the pulmonary artery.

Where tuberculosis supervenes in an already progressing consumption, it modifies the result and hastens the fatal issue.

Chronic tuberculosis can no longer be accepted as being in existence, since the elucidations concerning the origin of tubercle have taught that a greater part of the small, indurated nodules are dissolved in the broken-down inflammatory products. Where solitary tubercle or a very small crop primarily exists with or without a cheesy focus, its inevitable fate is to die, thus allowing no time for it to linger for more than a very limited period.

Persistent miliary tubercle must at the present day be denied.

Then we have certain *inflammatory lesions* of the lung, which, during the progress of the disease, are always combined, and, according as one or the other preponderates, we have separate forms of consumption. We may distinguish between those which occur *independently*, and run a slow course and are "chronic" from the beginning, and such as are the *remnants of past acute inflammations of the lung.*

Of independent forms we have :

Simple chronic indurative inflammation of the apex (chronic interstitial pneumonia) occurs in the alveolar and subpleural connective tissue.

Localized proliferation of peribronchial connective tissue, nearly always in connection with a cheesy node, which it endeavors to encapsulate. This form is independent only in not being a remnant.

Remnants of past acute inflammatory troubles, exudations which have undergone cheesy degeneration, so-called caseous

pneumonia. *Croupous pneumonia* likely furnishes no cheesy remnant, though this possibility is still believed in by some, and is chiefly based upon the fact that croupous pneumonia, as well as any of the other forms, is said to occur as an accompaniment to general diseases.

Catarrhal pneumonia. Negatived by Buhl and others; believed to be the source of cheesy products by Juergensen, the late Niemeyer, and others. The question here turns upon the products of such inflammations, the secretion, and the desquamated epithelium, which do undergo cheesy metamorphosis. If the shedding of epithelium is so marked (for other characteristics mentioned by Buhl, see chapter on Desquamative Pneumonia) as to call for a separate picture of disease and to be termed "desquamative pneumonia," then the question solves itself.

I believe that the products of such inflammations of the lung as accompany general diseases are only more prone to cheesy degeneration, because in independent acute inflammations the patient does not suffer from the combined effects of convalescence from a general disease and a fresh local trouble.

In this connection, I must again insist that, in the absence of any connecting link between general diseases and the subsequent occurrence of consumption, the most plausible theory to account for the predisposition of the patient and for his anæmic condition is:

From the moment of rise of temperature until normal degrees are regained, the formation of red blood-corpuscles and fibrine ceases. The consequence is that the waste will be greater than the supply.

The products of *hæmoptysis*, of *pleurisy*, of *purulent peribronchitis*, *embolic pneumonia* begetting anæmic necrosis, local *gangrene*, and *abscess*.

Now, either one of these three great factors—*tubercle*, *connective tissue hyperplasia*, *cheesy centres*—may exist in the lung of an individual without causing appreciable symptoms, thereby escaping diagnosis. It is when their constituents begin to break down, and they commence to be interwoven together, that they cause catarrh of the mucous membrane and râles may be heard.

On dissection it is ofttimes possible to determine which of the three conditions kept the lead during lifetime.

As a rule, we may say, when considering the likelihood of tubercle supervening in an existing chronic process, that that product of a cicatrization (whether of hyperplasia of connective tissue or cheesy node, or both) which has most stumps of the original pulmonary artery remaining in its periphery, and is the most indirectly nourished, is most apt to have tubercle supervening within it.

THERAPEUTICS.

Since you request it, and do not ask for more than "a general outline of therapeutics," I will add a few general hints as to what treatment should be based upon.

The two great factors in the treatment of consumption are *individualization* and *strict attention to detail.*

The indications for medical action should be evolved from the pathology, the exact knowledge of the course any given process is likely to take, which in turn implies equal exactness in diagnosis. It will not suffice to establish that your patient is a victim to consumption. He likely enough knows it himself, or at least has a strong premonition that such is the case.

Remember, also, that with all modern means of diagnosis on hand, we are unable to positively diagnose a strictly localized process at the apex before it is amenable to auscultation, and the symptoms then presenting themselves are those of an actively progressing trouble. No fault of ours, if we are so rarely consulted in the earliest stages. Nor of the patient's, whose attention is not called to it until a *persistent gastric catarrh*, with more or less fever, fixes his attention. Indeed this is by far the most frequent immediate cause of the retrograde changes which from thence take place in the apex.

But I was going to write of general therapeutics, and not of diagnosis, however tempting that field may be. What I wanted to say was, that the same exactness in individualizing in each separate case is stringently called for in therapeutics as in diagnosis. It is this strict individualization, too, which will guide you in deciding whether a patient should be without delay sent to an appropriate climate, under proper directions, or whether he can be treated at home. It is the loss of valuable time, entailed by first trying the home treatment, and, as a last resort, sending the patient to Florida, Nassau, Colorado, or Europe, which causes such bad results to be recorded, even where great skill has been exercised.

Close attention to detail in the carrying-out of a plan of treatment has always seemed to me to be the key-note to final

success. It is this which of late has made the treatment of diphtheria more successful. Authorities, like Prof. Jacobi of this city, have insisted upon this, and in no small measure contributed to the reduction of the rate of mortality.

The same attention and never-lagging exactness with which every detail is carried out is the secret of the success of Lister's antiseptic treatment of wounds. The failures are all recorded on the side of only partial adherence to a plan. Whatever is rational in any given line of treatment, cannot be lightly dispensed with without endangering the success of the whole.

The broad indications are these: if anomaly of constitution, hereditary influences are at work, of which scrofulosis is now again claimed to be a leading example, our first duty is to remove the constitutional tendency, anæmia, etc., in which branch Buchanan has of late excelled. Our best hold is no doubt appropriate climatic influences. When we consider how many localities, extensive as well as strictly localized, exist all over this globe in the temperate zone, it seems a simple duty to spot such regions, and make them accessible, as far as possible, to all classes of society. If careful search be made, I am satisfied there is not a State in this Union which cannot boast of some locality where the requisite conditions of temperature exist.

Here also I may mention the results obtained by Dr. Brehmer of Goerbersdorf in Silesia, by individualization and strict attention to detail.

Dr. B. claims that he has demonstrated that reduced atmospheric pressure (lessened pressure of the air-column) and a consequent low barometer, forms the basis for the cure of consumption; not because the patient inhales either less or more air than he does in the plains, but because the pressure of the atmospheric column is sufficiently reduced to allow him to inhale, at one inspiration, the *same quantity of air with less exertion of muscular force*, an important factor for a person with flabby heart and respiratory muscles. And the same exertion of strength required for respiration in the plains will beget proportionally *far greater and more rapid nutritive changes*, more especially as the out-door exercise involves more or less climbing and calls for increased exertion of the respiratory forces. Before such additional exertion is required of the patient, however, the other important factors of the prescribed course: food, drink, cleanliness, light, absence of

mental exertion, have fully prepared him for this additional task. I should add, what in reality is self-evident, that the air of the mountains is cleaner, free from impurities, and contains more ozone than the atmosphere of the plains.

The effect of breathing under low barometrical standing is, 1st. The pulse becomes accelerated, whereby—a. The blood is forced through the lungs, instead of stagnating there, as it did before, and—b. Brings to an immediate standstill the proliferation of connective tissue, shedding of epithelium and the formation of tubercle, which were induced and kept up by the slackened circulation and the consequently slow and imperfect nutritive changes.

2d. Uninterrupted frequency of pulse, heart-contractions which overcome peripheral impediments in the lung and elsewhere, begets normal hypertrophy of the ventricles, enlarges and strengthens the muscular element of the heart.

The "thinned air" treatment is supported by general or local derivation to the skin by means of the shower-bath (with some force, not long continued), mixed food, Hungarian wine, etc.

Now let us see what is accomplished by *inhalation of compressed air*, by means of any of the numerous pneumatic apparatus, or the "pneumatic cabinet" so-called.* Here also it is claimed that the pulse is accelerated and the propulsive power overcomes the impediments in the peripheral circulation. True, but it is not continuous and only practised for a limited period each day. What effects are produced? The patient inhales under increased pressure a large quantity of oxygen, *in a comparatively short space of time and with increased muscular exertion.* But it is claimed that in the beginning it is so regulated as to make the exertion gradual. Theoretically this may be supposed to increase the muscular elements in proportion to the increase in the pressure afterwards exercised, but practically it is not true. The factors against that presumption are too numerous: twenty-three hours and over of time-out of twenty-four in which the patient breathes ordinary air; lives under the same conditions of assimilation of food and drink; the same mental strain (sure to

* For a review of the different methods and apparatus for inhaling compressed air, to exhale into diluted air, etc., as well as the latest publications on this subject, I refer you to the last number of Schmidt's Jahrbücher (Jahrgang, 1878–No. 2).

exist as long as he remains at home); less direct action of the sun's rays than he would have in the mountains; impurities of air, such as dust, which are inseparable from the atmosphere of the common earth's surface. But above all we will find three strong contra-indications:

1st. Not even the daily breathing of compressed air for a *considerable period* will suffice to increase the muscular elements or the elasticity of the central propulsive powers.

2d. It is *always* with greater *voluntary muscular exertion* that the sittings are accomplished, and it is this which gives rise to temporary acceleration of the pulse.

3d. This increased muscular exertion is made, then, at a time when the patient does not feel within himself the power to exert himself, and the effort is, to say the least, lost.

4th. Supposing the effect of the inhalation to be increased and more forcible heart's contractions, overcoming of the sluggishness of the peripheral circulation, and consequent rapid nutritive changes, these nutritive changes (even though they do not accompany the inspiration of condensed air, but take place subsequently) are brought about too *suddenly and forcibly*, and without that evenness of uninterrupted continuity which alone can insure permanent nutritive changes.

Attention to the mechanism of respiration, derivation to the skin *in loco* or in the arm, occasional use of ice-bags, or warm applications covered by oiled silk, the local shower-bath with friction, injections into cavities and infiltrated tissue, inhalations, transfusion of blood and milk, the hypophosphites, our old friend cod-liver oil, or, what is often more easily digested, pure olive oil, together with such alkaloids as may be dictated by an emergency, are all therapeutic agents of great value in the hands of a conscientious physician who can rise above routine practice.